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THE INDUCTION OF SPLANCHNIC ANALGESIA 161
GASTON LABAT, M.D. ..... NEW YORK, N.Y.

SUBLINGUAL FIBROMA 187
JOHN J. MORTON, M.D. ..... NEW HAVEN, CONN.

ACUTE PANCREATITIS 193
CARL EGGERS, M.D. ..... NEW YORK, N.Y.

POST-OPERATIVE STRICTURE OF THE COMMON BILE DUCT 210
E. STARR JUDD, M.D. AND VERNE G. BURDEN, M.D. ..... ROCHESTER, MINN.

CHRONIC PARTIAL INTESTINAL OBSTRUCTION IN AN ADULT DUE TO ARRESTED DEVELOPMENT OF THE LARGE BOWEL 217
R. G. LECONTE, M.D., W. E. LEE, M.D. AND T. MCK. DOWNS, M.D. PHILA., PA.

HEMORRHAGIC ULCER OF MECKEL'S DIVERTICULUM 222
REGINALD H. JACKSON, M.D. ..... MADISON, WIS.

A METHOD OF HERNIOTOMY UTILIZING ONLY WHITE FASCIA 225
EDMUND ANDREWS, M.D. ..... CHICAGO, ILL.

THE JACK-KNIFE POSITION FOR PATIENTS AFTER OPERATIONS FOR ABDOMINAL HERNIA 239
LEIGH F. WATSON, M.D. ..... CHICAGO, ILL.

THREE THOUSAND CONSECUTIVE HERNIOTOMIES 242
BRADLEY L. COLEY, M.D. ..... NEW YORK, N.Y.

THE SURGICAL ASPECTS OF XANTHOMA TUMORS 256
EDWIN M. MILLER, M.D. ..... CHICAGO, ILL.

EFFECTS OF THE INTRAMUSCULAR INJECTION OF SODIUM CITRATE UPON BLEEDING 268
SAMUEL G. HIGGINS, M.D. AND DAVID FISHER, M.D. ..... MILWAUKEE, WIS.

INTRACRANIAL ARTERIO-VENOUS ANEURISM OR PULSATING EXOPHTHALMOS 272
CHARLES EDWARD LOCKE, JR., M.S., M.D. ..... SAN FRANCISCO, CAL.

BOOK REVIEWS: *Cotton*, Dislocations and Joint-fractures. *Horsley*, Operative Surgery. *Cabot*, Modern Urology 286

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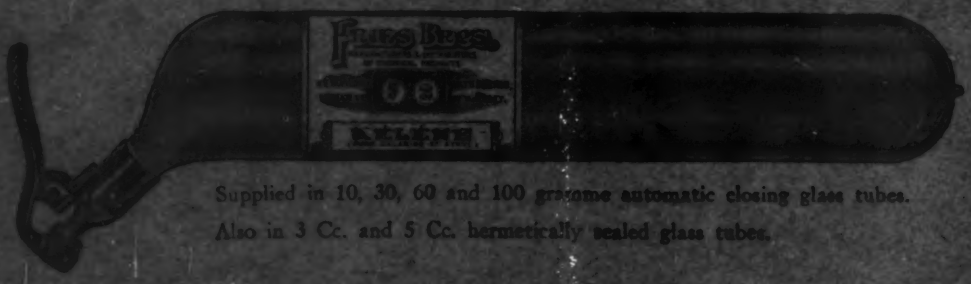

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ANNALS of SURGERY

VOL. LXXX AUGUST, 1924 No. 2

THE INDUCTION OF SPLANCHNIC ANALGESIA

FROM A CLINICAL AND ANATOMICAL STANDPOINT

BY GASTON LABAT, M.D.

OF NEW YORK, N. Y.

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IT CANNOT be proven by statistics that major abdominal operations performed under general narcosis are associated with greater dangers than when regional anæsthesia is utilized; nor can it be statistically determined whether or not the induction of regional anæsthesia involves greater risks than the administration of ether narcosis. It is a matter of opinion based on more or less justifiable attitudes and the personal equation of the operator plays a very important part in the achievement of the end-results. But it is well established that local anæsthetics, *when used judiciously,* limit their field of action to the anæsthetized area and leave practically undisturbed the functional activities of the organs on which life chiefly depends. That post-operative disturbances, which are mostly post-anæsthetic, are considerably minimized, morbidity and mortality greatly reduced when regional anæsthesia is the method employed, cannot be honestly denied. That there are cases in which general narcosis should absolutely be avoided is most certain, whatever be the attitude of the profession to the question of regional anæsthesia or general narcosis in abdominal surgery. It is chiefly on behalf of the bad-risk patients that splanchnic analgesia has been devised.

Review of Literature.—More than twenty-five years ago the first laparotomies for gastrostomy and gastro-enterostomy were performed under local anæsthesia. Local infiltration according to the method of Reclus or Schleich was the procedure used for anæsthetizing the abdominal wall. Manipulation of the deeper structures was rendered possible only by the administration of general narcosis; and this combined anæsthesia was, for a long time, considered the method of choice.

Braun's[3] method of anæsthetizing the abdominal wall was the first step toward widening the scope of local anæsthesia in major abdominal surgery. It gave a wider field of anæsthesia and better exposure of the viscera, as a result of considerable muscular relation.

Finsterer's[5, 6] "Mesenterial anæsthesia" mentioned at the German Congress of Scientists in 1912, was sufficient in a fairly good number of gastric operations, but failed to give satisfaction in extensive resections of the stomach which had to be completed under general narcosis.

Kappis'[12] method of inducing paravertebral anæsthesia was the next important development. It was based on the assumption that the sympathetic system was res-

ponsible for pain perception in the abdominal cavity and that the rami-communicantes transmitted the painful impulses to the central nervous system. Paravertebral anæsthesia of the upper abdominal organs was, however, abandoned by its author as soon as splanchnic analgesia was made possible by easier and quicker procedures.

In the winter of 1913-1914, Kappis [14, 15] began to induce regional anæsthesia for abdominal operations by local anæsthetization of the abdominal wall and injection of the area supplied by the splanchnic nerves and of the lumbar rami-communicantes (L^1 to L^3) proceeding from the dorsal aspect of the body. The first announcement of splanchnic analgesia was made by him at the German Surgical Congress of 1914.

In 1917, Wendling [43, 44] described a method of his own by which the area supplied by the splanchnic nerves was reached at the level of the cœliac plexus, the needle being introduced through the anterior abdominal wall, the left lobe of the liver and lesser omentum until its point came in contact with the vertebral column.

The year after (1918), Kappis [15] reported 200 operations, mostly stomach and gall-bladder, the majority of which had been performed since the early part of 1917.

The fifth edition of Braun's book (1919),[3] contains a description of its author's method of inducing local anæsthesia for gastric operations, by injecting the anæsthetic solution on both sides of the vertebral column through a point of entrance between the aorta and vena cava at the level of the first lumbar vertebra just above the pancreas; the injections being made after laparotomy.

In 1919, Naegeli [29, 30] used the posterior route of Kappis with a modified technic and published the results of his experiences with splanchnic analgesia which included 18 cases mostly stomach. Buhre [5] described the procedure divised by Braun and reported 33 cases of splanchnic analgesia induced after opening the abdominal cavity. Pauchet [25] used the same procedure. Denk [4] published his results with Kappis' technic. Preiss and Ritter [27, 28] reported 89 cases of splanchnic anæsthesia in 15 different types of operations (abdominal and renal). At the French Congress of Surgery held in Paris the same year. Marc Roussiel [40] announced that the method he used consisted in the injection of D^{10} and D^{11} or D^{11} and D^{12}, which was a true blocking of the splanchnic nerves, and that the anæsthesia thus produced was sufficient for all gastric operations.

In 1920, I [22] reported the first 12 operations performed in France under splanchnic analgesia according to Naegeli's technic which is a modification of Kappis', and introduced the method in this country.[8] The year after I published in England the results of my experience with 34 major abdominal operations also performed in France (Pauchet's Clinic, Paris) with the same technic. Hoffmann,[9] Khautz,[10] Paul [26] and others also contributed to the literature on the subject.

In 1922, Mirizzi [23] reported 26 cases with Kappis' method and 3 cases with Braun's method. Gandusio and Pototschnig [7] used Kappis' method in 18 cases; Quarella [29] in 12 cases.

Last year Metge [27] reported 112 operations following Kappis' technic exactly. Hustinx [11] added 119 cases to those already on record using Braun's technic as well as Kappis'. Finsterer reported, in one of his lectures at University and Bellevue Hospital Medical College, N. Y., 566 gastric resections performed under splanchnic anæsthesia according to Braun's technic. As far as I know this is the largest series.

Since my last publication in the *British Journal of Surgery* I have added a few more cases to my credit. The two gastric resections reported in this paper are among my latest demonstrations of splanchnic analgesia in major abdominal surgery. I am greatly indebted to Drs. Charles W. Walker and Louis F. Lange for their patients' histories and personal post-operative observations.

Abdominal Sensibility to Pain.—Our present knowledge of the transmission of painful stimuli from the abdominal cavity and viscera to the central nervous system is largely due to Neumann [31, 32, 33] and Kappis,[13, 16] who arrived at practically the same conclusions based on animal experimentation.

Until the year 1911, nothing definite had been published on the sensibility of the abdominal organs and Lennander's [25, 26] opinion was still prevailing among the great majority of surgeons, *viz.*, that the viscera are insensitive and that the parietal peritoneum is the only pain-receptive structure of the abdominal cavity. Clinical observers were and are still agreed that the viscera are insensitive to painful stimuli, and that pain commences as soon as tractions are exerted on the viscera or their pedicles. Exploratory manœuvres during which the bowels are displaced are also associated with pain whose intensity varies with the susceptibility of the patient and the nature of the manipulations.

In the summer of 1912, Kappis,[13, 16] conducted a number of experiments on the dog with a view to answering the question of sensibility of the abdominal organs. His findings were that sensory nerves accompany the blood-vessels going to the viscera, but that their number is relatively small considering the extensive territory which they supply; for this reason the nerves lose their power of transmitting pain before they enter the organs. "It may be," says Kappis, "that the pain transmitting fibres cease before the entrance of the nerves into the organs. It may be that they are not in sufficient number and strength to allow perception of the sense of pain. At any rate the abdominal viscera may be regarded as practically without sensation. But this does not say that the visceral peritoneum and its contents lack every power of pain perception. On the contrary, at those points where nerves are present in sufficient number and strength, and that is practically speaking the larger, medium, or at least not the very small vessels, a distinct pain perception may be proved to exist under all forms of stimulation.

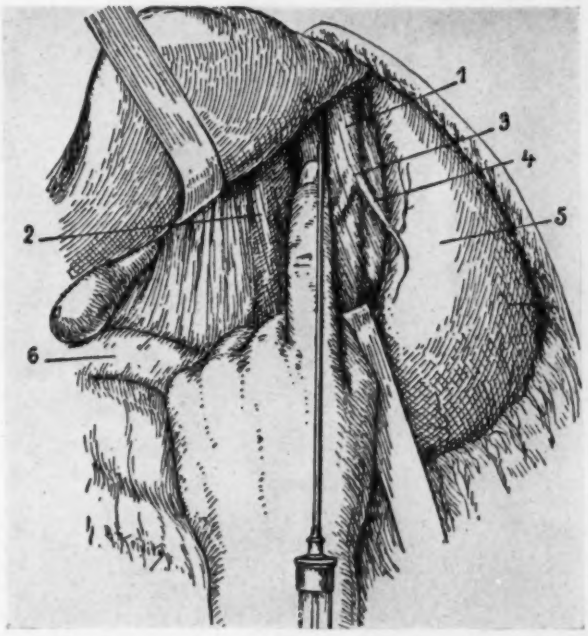

FIG. 1.—Splanchnic analgesia by the anterior route. (Braun *in* Finsterer.) Left lobe of liver retracted upward, stomach drawn down, through which the small omentum is stretched. 1, aorta; 2, vena cava; 3, cœliac axis; 4, arteria gastrica sinistra; 5, stomach; 6, duodenum.

On account of this in the visceral peritoneum of the dog, those parts are pain-perceptive or at least not insensitive under proper stimuli which carry the larger nerves and vessels; in other words the blood-vessel-carrying portion

of the mesentery, the small omentum, the attachment of the greater omentum to the stomach and naturally the great vessels themselves". Neumann,[31] Kappis[13] and Hoffmann[10] found that in dogs section of the splanchnic nerves abolished pain in the stomach, intestines and bile-ducts.

In 1913, on the strength of these findings, Kappis began to induce regional anæsthesia for abdominal operations by injecting the splanchnic nerves as they enter the semilunar ganglia. His results aroused the interest of the other men already engaged in abdominal surgery under regional anæsthesia and suggested other means of approach to the splanchnic nerves and their collaterals.

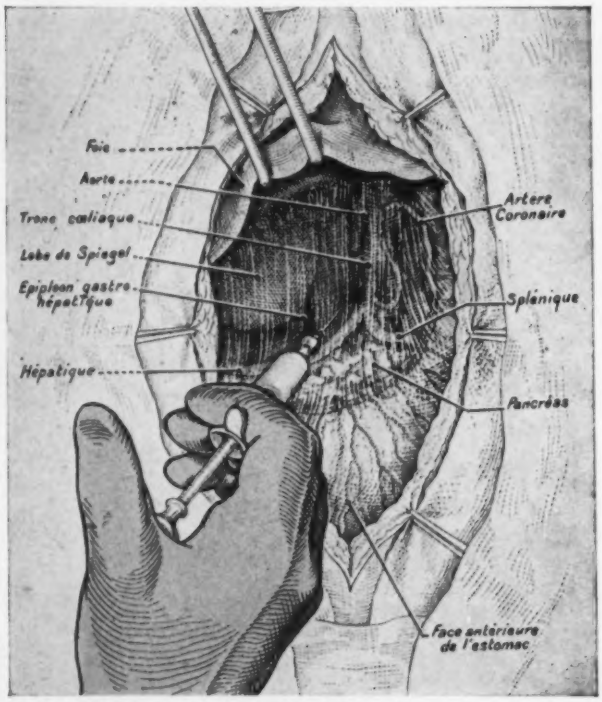

FIG. 2.—Surgical anæsthesia of the solar plexus. The liver is retracted upward; the upper margin of the pancreas is defined by palpation which at the same time locates the aorta by its beats; the needle is introduced to the right of the aorta and injection is made of 20 c.c. of a 1 per cent. solution of neocaine. (Pauchet.)

Methods of Inducing Splanchnic Analgesia.—There are three methods of inducing splanchnic analgesia: (a) Kappis',[15, 16, 17, 18] (b) Braun's,[2] and (c) Wendling's;[43, 44] but there are only two routes of approach, *viz.*, posterior and anterior. These methods aim at depositing the solution in close proximity to the splanchnic nerves soon after their passage from the thorax into the abdominal cavity, at the point where they reach the semilunar ganglia. Kappis uses the posterior route of approach on both sides of the vertebral column; Braun and Wendling proceed from the front. While Wendling attempts to reach the region without opening the abdominal cavity, Braun exposes the field of injection by a median laparotomy.

Kappis introduces his needle on the lower border of the 12th rib, at a point 7 cm. distant from the middle line of the back, in a direction ventralward making an angle of 30 degrees with the median plane of the body and injects the solution as soon as the point of the needle has reached the union of the lateral and anterior surfaces of the body of the vertebra. Other injections are also made up and down the side of the vertebral column "in order to make sure to reach the area at which the splanchnic nerves enter the abdominal cavity as well as their ramifications".

FIG. 3.—The lesser abdominal cavity. Stomach resected between pyloric end and a line passing a few centimetres below the cardia. Peritoneum and nerve plexuses removed leaving the semilunar ganglia on each side of the cœliac axis. Blood-vessels, crura of the diaphragm, pancreas and parietal attachment of the transverse mesocolon exposed after large resection of both costal margins and retraction of the left lobe of the liver upward.

Braun opens the abdomen on the middle line, retracts the left lobe of the liver upward, pulls the stomach downward and a little to the left, locates the aorta above the pancreas and injects the retroperitoneal tissue between the aorta and the vena cava after pushing the aorta to the left with his palpating finger.

Wendling introduces his needle to the left of the tip of the xiphisternum and attempts to reach Braun's site of injection by transfixing the left lobe of the liver and the lesser omentum.

Of these three methods, Wendling's has been discarded—I should say not even attempted on patients—because of its dangers. It has been repeatedly tried on the cadaver and found to be very unsafe, to say the least, considering the anatomy of the region. To be convinced of the merits of Wendling's method one should read Kotzareff's[20] comments following Sourdat's[42] publication on the subject.

Braun's method is safer and simpler than Wendling's, since the injection is made after opening the abdomen and under the control of a guiding finger and the eye sometimes. But the manœuvres associated with this procedure are painful, *viz.*, raising the left lobe of the liver, pulling the stomach, palpating and retracting the aorta. Certain patients cannot stand them unless they be placed in a condition of twilight sleep by heavy doses of morphin and scopolamin. A contracted, highly situated, large or adherent stomach is much in the way and renders the technic absolutely impracticable. A right paramedian laparotomy is out of line with the site of injection and precludes the use of the method. A left rectus incision makes the retraction of the liver very difficult and rather insufficient.

Mirizzi[28] found that the injection of the splanchnic nerves according to Braun's method was always followed by infiltration of the desired area in the cadaver, but when applied to patients the result was satisfactory in only one case out of three. Even under favorable conditions failures are actually experienced with Braun's method which involves not only the difficulty of differentiating the beats of the aorta from those of the cœliac axis, but the possibility of puncturing the largest veins, *viz.*, vena cava and vena porta (Fig. 3) in which case Finsterer advises against further attempts to inject.

Kappis' method is applicable to any case, whatever be the type of laparotomy and the pathological condition of the upper abdomen. Modifications based on individual experience have been described (Naegeli,[29, 30] Labat [22, 23, 24]), their main object being to strengthen the weak points of the originator's technic and raise the danger signal at the approach of inexperienced hands. Thus in changing the angle advised by Kappis from 30 degrees to 45 degrees we have cautioned against passing the needle too far away from the vertebral column into the important neighboring structures; in mentioning and illustrating the level at which the needle first takes contact with the vertebral column we call attention to the intervertebral foramen which must be left behind (Fig. 4). It is obvious that if the needle were introduced carelessly at an angle less than 30 degrees it would leave the paraspinal muscular struct-

ures, pass through the fatty capsule of the kidney and puncture the renal pedicle, or not infrequently transfix the kidney, as shown by some of my cross sections. The needle would likewise come in contact with the left renal vein (Billet and Laborde,[1] Quarella[39]), the vena cava or the aorta were it introduced deep enough.

On the other hand, if the angle of 45 degrees which the needle makes with the sagittal plane of the body in its first thrust through the soft structures of the

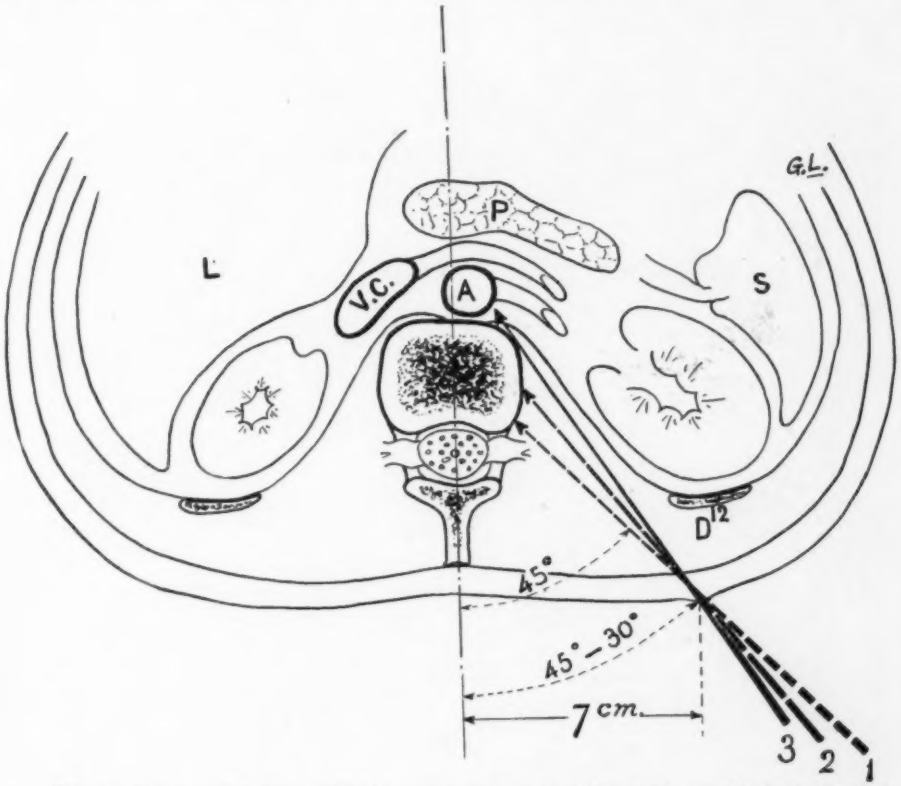

FIG. 4.—Diagrammatic illustration of a cross-section of the body passing through the first lumbar vertebra at the level of puncture and injection. Arrows 1, 2 and 3 give in succession the manner of reaching the solar plexus with safety.

back were increased to any extent, the point of the needle would occasionally reach the intervertebral foramen which is the only dangerous point backward. It would then either enter the spinal canal or induce paræsthesias before entering it. But the second step of our technic would correct the error of direction, since the needle must be partially withdrawn and reintroduced at a smaller angle until its point is felt gliding along the body of the vertebra. There is therefore no risk of making an intraspinal injection, so long as the angle at the time of injection is less than 45 degrees, no more bone felt in front of the needle, the point of the needle 10 to 12 cm. deep and no paræsthesias induced at that depth. During the whole procedure the needle must be kept strictly in the transverse plane of the body passing through the site of puncture.

To inject intraspinally or throw the anæsthetic solution into the blood stream, one must either experiment on patients without having carefully studied the method and familiarized himself with its attendant circumstances or use very poor technic.

Judging from the literature and according to my experience. Kappis'

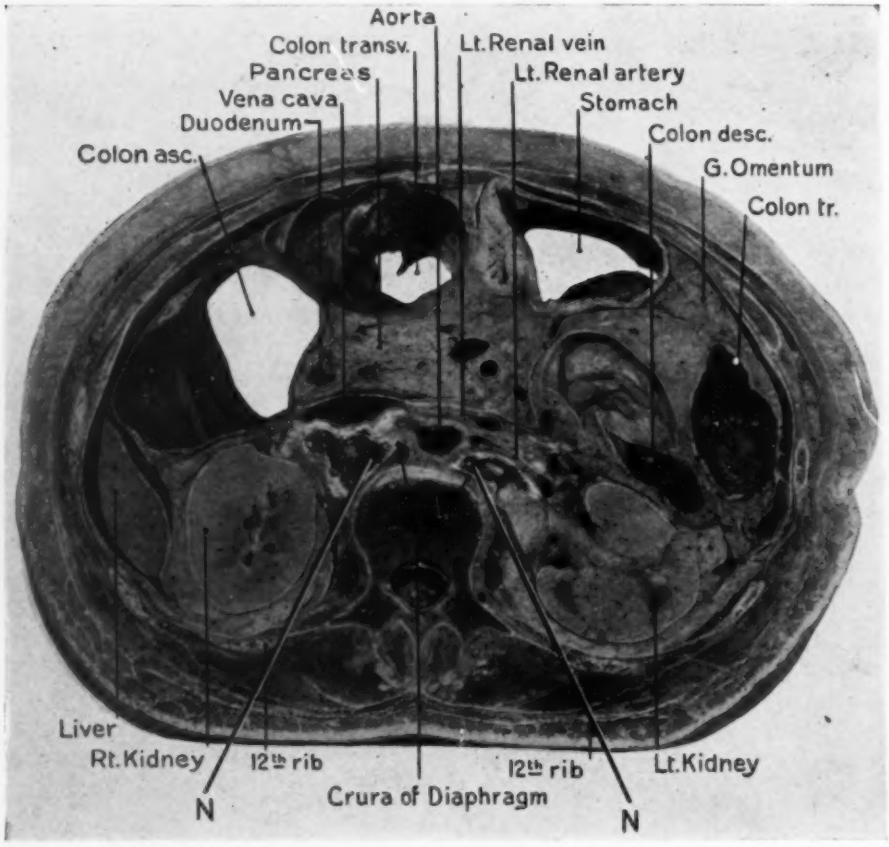

FIG. 5.—Cross-section of female body (upper segment) through the usual sites of puncture on the lower border of the 12th rib, 7 cm. distant from the middle line of the back, illustrating the zones of infiltration of minium-starch (40 c.c. on each side) injected immediately before embalming with formalin. The section passed through the lower part of the body of L^1 and just above its spinous process. Formalin caused a contraction of the soft parts with consequent reduction of the costo-vertebral angle, thus altering the relation of the site of puncture to the 12th rib. Note the left renal pedicle at that level.

posterior route is the simplest and safest route of approach to the splanchnic area, whatever be the attitude of those who are afraid of inserting a needle through the musculature of the back, but boldly seek in the highly vascular territory of the lesser cavity (Fig. 3) a safe passage for the needle and the correct spot at which the anæsthetic solution must be deposited. Of course, the induction of splanchnic analgesia is a major procedure which requires skill and experience, just the same as gastrectomy, mostly for which it is given, is a major operation which the novice does not tackle with impunity. But in the hands of men already familiar with regional anæsthesia the injection

of the splanchnic nerves by the posterior route is as safe as a paravertebral block.

I have repeatedly made injections of methylene blue in the cadaver and have never found the solution in one of the blood-vessels of the region. Safety is greater in the living being, because one can tell at any time whether or not a blood-vessel has been punctured. I have injected minium-starch in cadavers before embalming them and made cross-sections two or three weeks

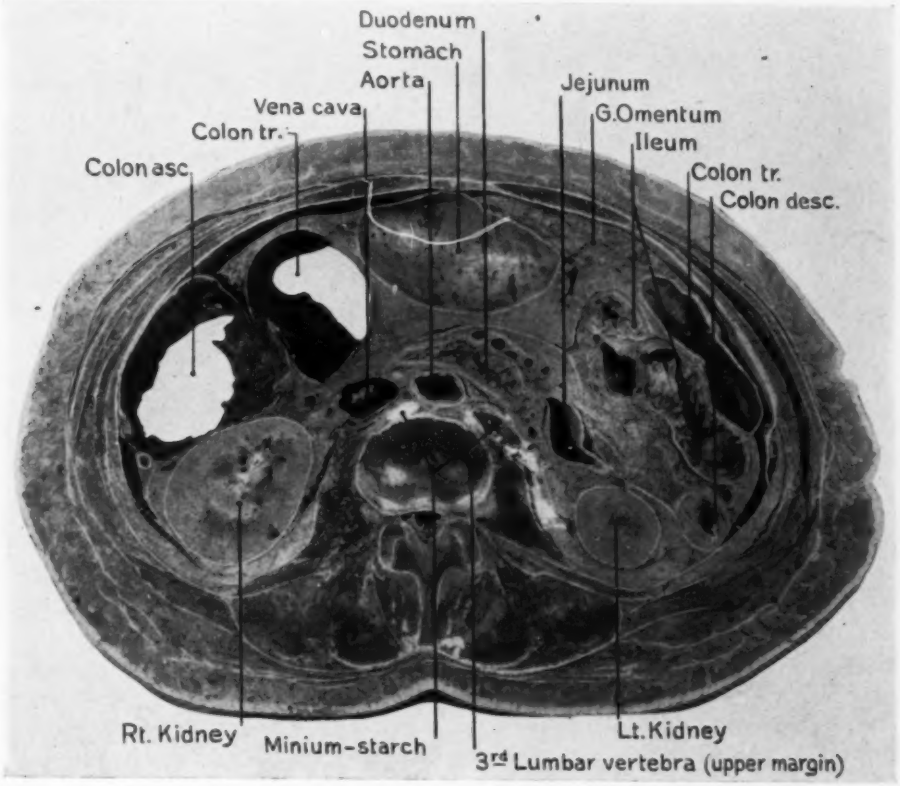

FIG. 6.—Cross-section (upper segment) of the same body as Fig. 3 passing through the upper margin of the body of L^3 showing very little of the injected minium-starch at that level, which is a proof that the bulk of the solution stays over L^1 and L^2.

later. The disposition of the starch in these sections is very instructive and conclusive (Figs. 5 and 6). On the right side the vena cava is pushed frontward by the injected fluid which distributes itself up and down along the flank of the vertebral column and covers a space considerably greater than that occupied by the splanchnic nerves, semilunar ganglia and rami-communicantes of the first three lumbar nerves. On the left side the injected fluid did not pass in front of the aorta but nevertheless reached the goal. Methylene blue solution spreads over such an extensive area in the cadaver that one would believe it possible to attempt any major abdominal operation after making only one injection on each side of the vertebral column. In the living being the retroperitoneal tissue is loose and easily distended by the anæsthetic

solution which in the majority of cases raises the pancreas from its seat; but novocaine solutions do not seem to diffuse as rapidly and extensively in live tissues as methylene blue in the dead body. Splanchnic analgesia is applicable more to the surgery of the upper abdominal organs and kidney than to that of the other parts of the abdomen, at least as far as our present experience goes.

Contributing Factors of Success.—Successful anæsthetization of the splanchnic nerves and their ramifications by the posterior route depends on many factors among which are: (a) general visualization of the anatomic structures of the region, from the superficial layers of the back to the constituent elements of the posterior wall of the lesser abdominal cavity; (b) tactile sense, delicate enough to transmit to the brain a fairly accurate knowledge of the structures with which the point of the needle comes in contact, in order to have a more precise visualization of the essential features closely connected with the deep landmarks; and (c) accuracy in taking the superficial landmarks. This last but not least factor we are now particularly interested in.

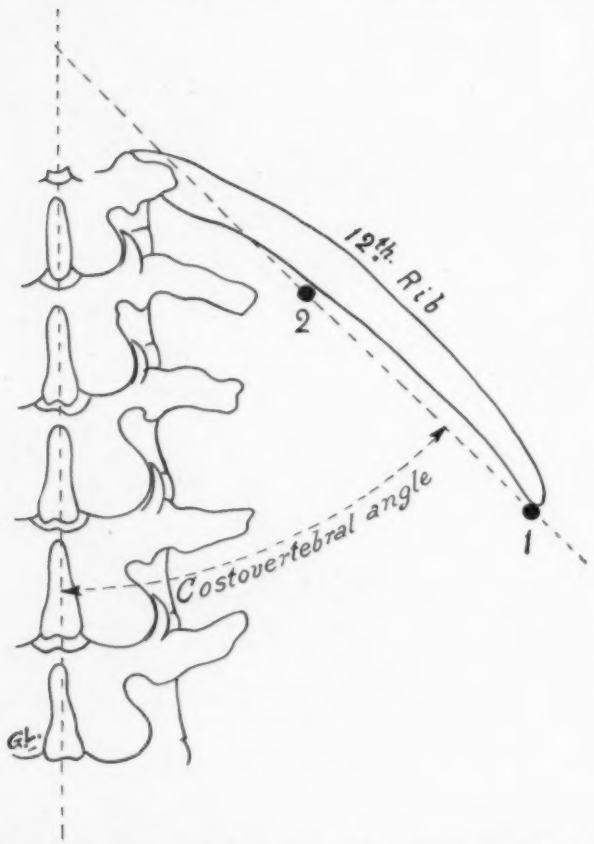

FIG. 7.—Wheals 1 and 2 are raised at the extreme palpable points of the lower border of the 12th rib which is defined by palpation from below upward and inward. The straight line passing through these two wheals marks with sufficient accuracy the direction of the 12th rib. The angle contained between this line and the middle line of the back is the costovertebral angle.

With the exception of Buhre's,[3] not a single report hitherto published has failed to register a certain percentage of cases in which it was necessary to complete the anæsthesia by some other means (local infiltration or general narcosis); and what is more surprising is that individual technical experience does not seem to constitute an absolute guarantee against failures. Greatly interested in the cause resulting in such failures, I made a scrupulous analysis of the attendant circumstances and came to the conclusion that variations in

the anatomy of each individual were chiefly to be blamed when the injection technic was correct and the skill of the operator sufficiently great.

The ease with which the 12th rib is defined varies chiefly with the weight of the patient, the degree of relaxation of the musculature in its immediate neighborhood and the distortion of the spine due to position. The direction of the 12th rib can be accurately traced on the surface of the skin only when a sufficient length of its distal end is made salient by deep but gentle pressure applied particularly along its lower margin. The straight line

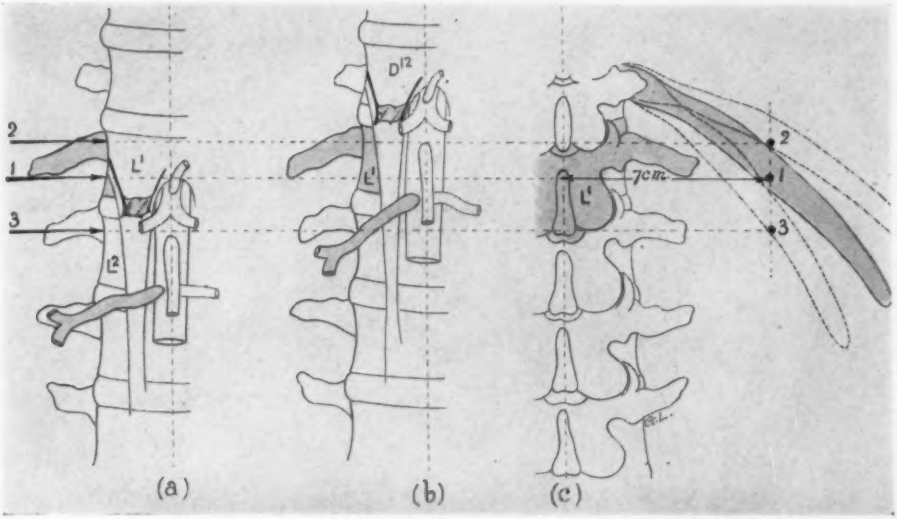

FIG. 8.—Diagrammatic illustration of actual results obtained with a constant site of puncture. While the costovertebral angle varies (c), note the levels of injection in positions (a) and (b) of the semilunar ganglia. 1, on normal rib is pretty good in (b), too high in (a). 2, on rib with costovertebral angle greater than normal is good in (b), too high in (a). 3, on rib with reduced costovertebral angle is good in (a), too low in (b).

obtained by joining the extreme palpable points (Fig. 7) may then be prolonged upward and inward, so that a point may be taken on it 7 cm. distant from the middle line of the back, according to the technic established for the posterior route.

Costovertebral Angle.—The direction which the 12th rib makes with the middle line of the back (last costovertebral angle) varies in a certain percentage of cases. Of the 52 dead bodies, mostly males, examined recently 37 presented an angle of 45 degrees and slightly greater; 10 an angle of 40 degrees; 2 an angle less than 40 degrees; 1 an angle of 55 degrees; 1 an angle of 60 degrees; 1 an angle of 70 degrees. Variations exist in both sexes, as shown by clinical observations, and they are in many instances, I believe, the cause of partial or total failure to obtain anæsthesia.

Since the distance of 7 cm. from the middle line of the back remains constant, variations in the costovertebral angle evidently result in variations in the level of the site of puncture and that of injection. Thus instead of reaching the body of the first lumbar vertebra the point of the needle glides along the body of the second lumbar vertebra if the angle is small. It stays

behind the pillars of the diaphragm if introduced at a higher level, as the result of measurements taken on a 12th rib which makes an abnormally great angle with the vertebral column (Fig. 8). It is therefore necessary to introduce in the basic principles of technic a coefficient of correction of the last costovertebral angle, so that the level of the site of injection may remain constant.

Semilunar Ganglia and Renal Pedicles.—The semilunar ganglia which are the targets of the needle lie either on the disk between D^{12} and L^1, or on that uniting L^1 and L^2 from which they are separated by the crura of the diaphragm. They may rest on the upper margin of L^1 (Fig. 9) or be situated on the upper portion of L^2. Differences of level on either side have no technical importance.

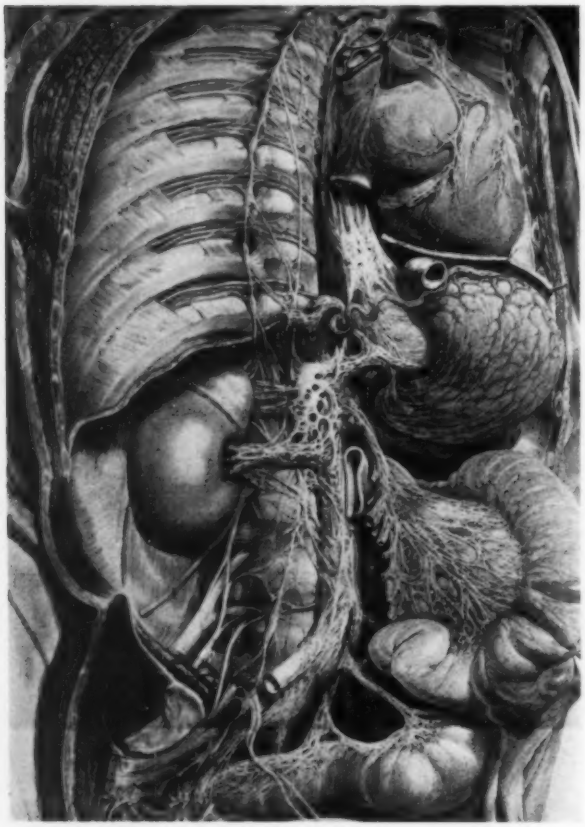

FIG. 9.—The upper abdominal sympathetic system showing the right semilunar ganglion at the lower margin of D^{12} and the right renal artery crossing the upper margin of L^2. (Bourgery et Jacob, Anatomie de l'Homme.)

In 12 bodies examined, the right and left semilunar ganglia were found at approximately the same level on both sides. They lie on either side of the cœliac axis and, in most cases, somewhat lower than its origin but higher than that of the superior mesenteric artery. The left semilunar ganglion was more ventralward than the right which was partly hidden by the vena cava. In 3 cases the ganglia were at the disk between D^{12} and L^1; in 3 cases at the upper third of L^1; in 1 case at the middle of L^1; and in 5 cases at the disk uniting L^1 and L^2. In 1 case there were only 11 ribs present with a very short rib (2 cm. from its costotransverse articulation) attached to the vertebra below, which was the first lumbar vertebra. In this case the semilunar ganglia were at the middle third of L^1 and the renal pedicles at the disk between L^1 and L^2 (Fig. 10). There were 2 cases of very short 12th rib (5 cm. and 10 cm. measured from the middle line of the back). The former could not be palpated while the presence of the latter was revealed only by tactile

TABLE I.

Table Showing Results of Examination of Twelve Dead Bodies with Special Reference to the Levels Occupied by the Semilunar Ganglia and Renal Pedicles.

No. of dead bodies examined	12th rib				S. L. Ganglia		Cœliac Axis	Renal pedicles				Relation of site of puncture to	
	Length cm.	Angle	Pleura		Right	Left		Right		Left		Spinous process	Vertebra
			R.	L.				A.	V.	A.	V.		
1	17	45	0	4 cm.	$L^1 - L^2$	$L^1 - L^2$	$D^{12} - L^1$	$L^2 - L^3$	$L^2 - L^3$	$L^2 - L^3$ (trifle higher)	$L^2 - L^3$	L^1 up	$L^1 - L^2$
2	20	45	7 cm.	4 cm.	$L^1 - L^2$	$L^1 - L^2$	L^1 low	L^2	L^2	$L^2 - L^3$	$L^2 - L^3$	L^1 up	$L^1 - L^2$
3	14	45	7 cm.	5 cm.	$L^1 - L^2$	$L^1 - L^2$	$L^1 - L^2$	$L^2 - L^3$	$L^2 - L^3$	$L^2 - L^3$ (trifle higher)	$L^2 - L^3$	L^1 up	$L^1 - L^2$
4	16	40	5 cm.	5 cm.	$D^{12} - L^1$	$D^{12} - L^1$	$D^{12} - L^1$	$L^1 - L^2$	L^2	$L^1 - L^2$	L^2	L^1 low	L^2, low. than renal pedicles.
5	15	45	7·5 cm.	6 cm.	L^1 up	L^1 up	L^1 up	$L^1 - L^2$	$L^1 - L^2$	L^1 up	L^1 up	L^1 low	Body of L^2
6	18	45	9 cm.	5 cm.	$L^1 - L^2$	$L^1 - L^2$	$L^1 - L^2$	L^2 low	$L^2 - L^3$	$L^2 - L^3$	L^3 up	L^1 low	Body of L^2
7	18	45	6 cm.	4 cm.	$D^{12} - L^1$	$D^{12} - L^1$	$D^{12} - L^1$	$L^1 - L^2$	$L^1 - L^2$	L^1 low	L^1 low	L^1 low	$L^1 - L^2$
8*	20	55	4 cm.	0	L^1	L^1	L^1	$L^1 - L^2$	$L^1 - L^2$	$L^1 - L^2$	$L^1 - L^2$	L^1 up	Body of L^1
9	10	60	0	0	L^1 up	L^1 up	L^1 up	L^2 up	L^2 up	L^2	L^2	L^1 up	L^1 low.
10	5	70	0	0	$D^{12} - L^1$	$D^{12} - L^1$	$D^{12} - L^1$	$L^1 - L^2$	$L^1 - L^2$	$L^1 - L^2$	$L^1 - L^2$	L^1 up†	L^1 low.
11	18	40	9 cm.	5 cm.	$L^1 - L^2$	$L^1 - L^2$	L^1	$L^2 - L^3$	$L^2 - L^3$	$L^2 - L^3$ (trifle higher)	$L^2 - L^3$	L^2 up	L^2 low.
12	18	45	7 cm.	5 cm.	L^1 up	L^1 up	L^1 up	L^2 up	L^2 up	L^2	L^2	L^1 up	L^1 low.

(*) Eleven ribs only, the 12th being about 2 cm. and articulated with L^1 measurements were taken as if the 12th rib had been replaced by the 11th.
(†) Puncture made on lower border of 11th rib, the 12th rib being impalpable.
NOTE.—L^1, L^2, L^3: first, second, third lumbar vertebra, except in column 13 where L^1, L^2 refer to the spinous process of the first and second lumbar vertebræ; L^1–L^2: disk between first and second lumbar vertebræ; L^1 up: upper extremity, margin or portion of L^1; L^1 low.; lower extremity, margin or portion of L^1.

sense at its distal end. The costovertebral angle measured 60 degrees and 70 degrees respectively.

The *renal pedicles* (artery and vein) generally occupied one of the two following levels: disk between L^1 and L^2 (Figs. 10 and 11); disk between L^2 and L^3. They may have their origin slightly higher (as in Fig. 5) which is not in our series of 12, or lower than those disks, sometimes in front of the body of L^2; but I have never seen them beyond these extreme limits. They ordinarily slant gently downward and outward, when highly situated,

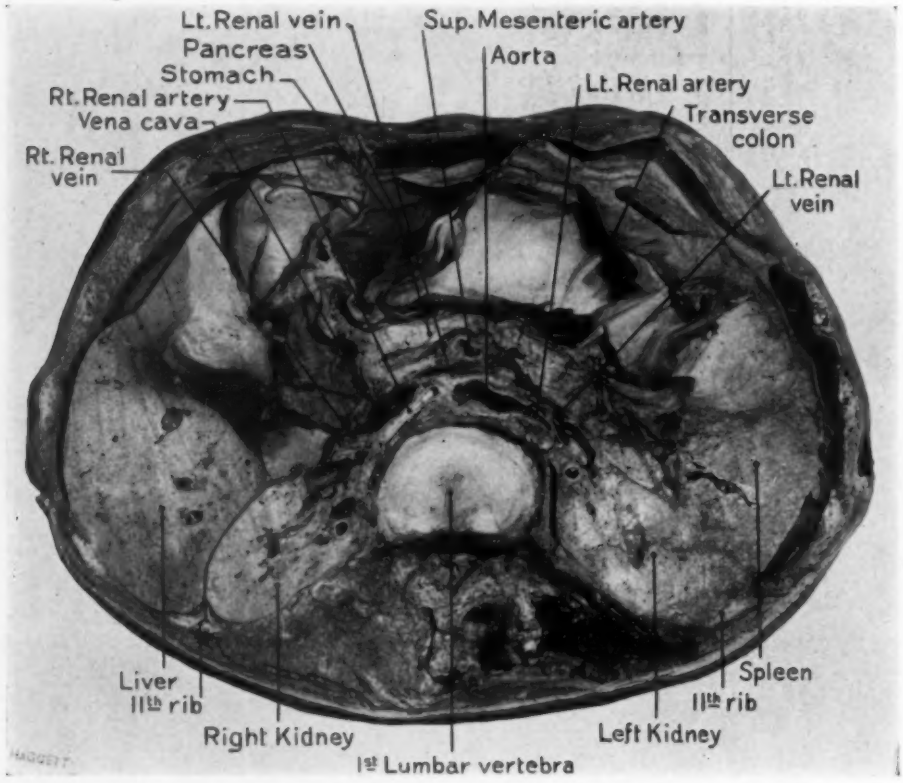

FIG. 10.—Cross-section passing through the intervertebral disk between L^1 and L^2 showing the renal pedicles at that level. This body had only 11 ribs (No. 8 of Table). The section was made through the usual site of puncture taken on the 11th rib which was thought to be the 12th.

especially on the right side. The artery is behind the vein which is a trifle higher or lower than the artery. The renal veins are very large, the left one crosses the anterior aspect of the vertebral column almost transversely. Differences of level between the two pedicles are of little significance as far as the danger of puncturing the left renal vein is concerned.

In the aforementioned series of 12 bodies (see Table I) the renal pedicles were found to be at the disk between L^1 and L^2 (Figs. 10 and 11) in 5 cases, which were those in which the semilunar ganglia were at the intervertebral disk between D^{12} and L^1 or in its immediate vicinity. These blood-vessels, especially on the left side, lay on the disk uniting L^2 and L^3 in 5 cases, and

this was when the ganglia stood at the disk between L^1 and L^2. They crossed L^2 in 2 cases in which the ganglia were at the upper third of L^1.

Coefficient of Correction.—Generally speaking the semilunar ganglia occupied a high level (D^{12} - L^1) as many times as they were found to be low (L^1 - L^2), and variations in the level of the renal pedicles occurred in an equal number of cases.

If it is not wise to conclude from the examination of 12 bodies, one thing seems so far demonstrated, that is, the full height of one vertebra between

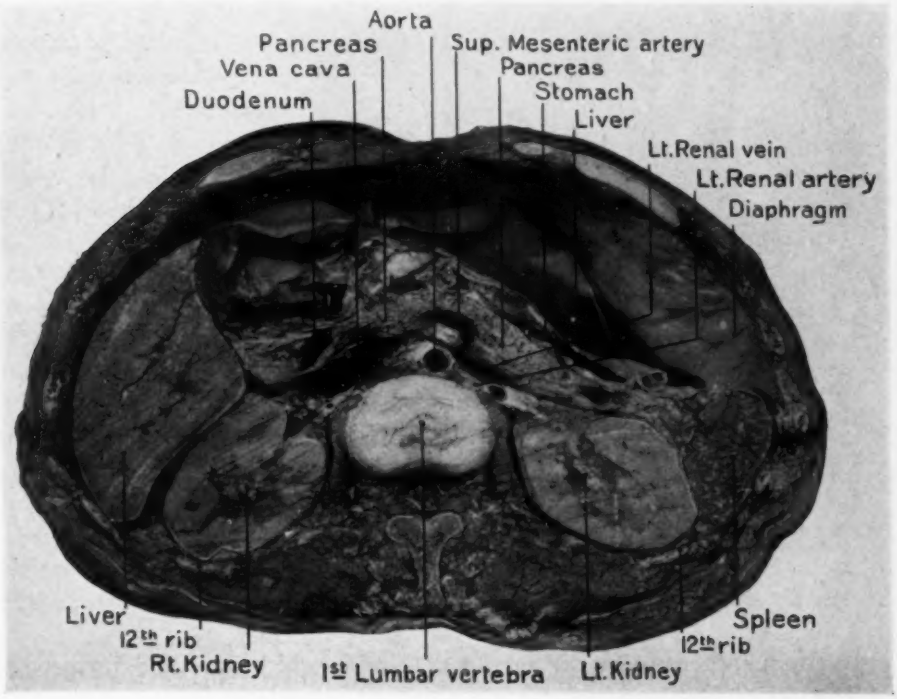

FIG. 11.—Cross-section passing through the intervertebral disk between L^1 and L^2 showing the left renal pedicle (artery and vein) and right renal artery at that level. The right renal vein was in the lower segment.

the solar ganglia and the renal pedicles. *That vertebra may be the 1st lumbar (Fig. 8, b) or the 2nd lumbar (Fig. 8, a)*, irrespective of variations in the costovertebral angle. It therefore seems logical to make the injection on the flank of L^1; so that, if the ganglia are high situated the anæsthetic fluid will be deposited just below them, if they occupy a lower level the splanchnic nerves will be reached just as they approach the ganglia.

If the solution is deposited on the lateral aspect of L^2, distribution takes place more along the renal pedicles, below the pancreas and toward the lower abdomen than in the direction of the semilunar ganglia; the centre of pressure of the injected fluid being too remote from its point of destination. If the needle is inserted higher than L^1 the injection is made partly behind the diaphragm and serves no purpose. It is therefore *on the lower margin of the body of the first lumbar vertebra,* that the anæsthetic fluid must be deposited in

order to produce its maximum effect, if only one injection is made on each side, as was the case in the two patients mentioned in this paper.

The body of the first lumbar vertebra can be fairly accurately determined from the surface of the skin by the relation which it bears to its own spinous process. The horizontal line passing through the upper extremity of the prominence marking the spinous process of L^1 generally passes across its body (middle or lower third). *The coefficient of correction is thus found to be the spinous process of* L^1; if the point taken on (not below) the lower margin of the 12th rib, 7 cm. distant from the middle line of the back, is not opposite the upper extremity of L^1 owing to variations in the costo-vertebral angle, it should be raised or lowered as the case may demand. *The shaft of the needle must be kept strictly in the transverse plane of the body passing through the site of puncture.* Distortion of the spine due to position has no influence on this correction of level. A very short impalpable 12th rib (5-6 cm.) and the presence of 11 ribs are the only abnormalities which tend to make the results somewhat doubtful.

FIG. 12.—Splanchnic analgesia by two injections from the same site of puncture on the lower border of the 12th rib, opposite the upper extremity of the spinous process of L^1. (Photographed from Bourgery and Jacob, Anatomy.) The solution is deposited above and below the disk uniting L^1 and L^2.

In a certain percentage of cases, *viz.*, those in which the ganglia occupy a low level, one injection on each side of the vertebral column is not followed by complete anæsthesia. It is therefore recommended to inject in every case from 25 to 35 c.c. of the 0.5 per cent. neocaine solution at the level of L^1 and from 20 to 25 c.c. of the 0.5 per cent. of the same solution on L^2 just below the intervertebral disk between L^1 and L^2, using the same site of puncture opposite the upper extremity of the spinous process of L^1 and giving to the needle the required direction downward (Fig. 12). The solution then distributes itself over a sufficiently wide area and not only reaches the semilunar ganglia wherever they may be, but soaks the spinal nerve plexuses of the posterior abdominal wall in the region exposed to direct operative manipulation.

Pleura.—The costodiaphragmatic sinuses were also examined with a view to investigating their relationship to the site of puncture and to the path of the needle, especially when there are only 11 ribs present and when the 12th rib is so short as to be misleading. The tabulated results show that in only 2 cases out of 12 did the costodiaphragmatic sinus on the right side cross the lower border of the 12th rib at a distance of 9 cm. from the middle line of

the back; its level near the spine being the transverse process of L^1 which is ordinarily just above the plane in which the needle is introduced. In body No. 8 reference is made to the 11th rib taken as the 12th rib. It was thus brought out that the needle, passed through a site of puncture on the lower border of the 12th or the 11th rib (in the absence of the 12th) and inserted in the transverse plane of the body, does not cross the costodiaphragmatic sinuses. Even if it did there would be no harm, since the solution is deposited at considerable distances from that portion of the pleural reflection. If a short 12th rib is overlooked and the puncture made with the 11th rib as landmark the needle may pass through the pleural reflection (Fig. 13), though in body No. 10 the pleura stopped higher than the transverse plane passing through the site of puncture taken on the 11th rib. It seems also that there is very little chance, if any, of traversing the costodiaphragmatic sinus on the left side. Puncturing the lung is out of the question.

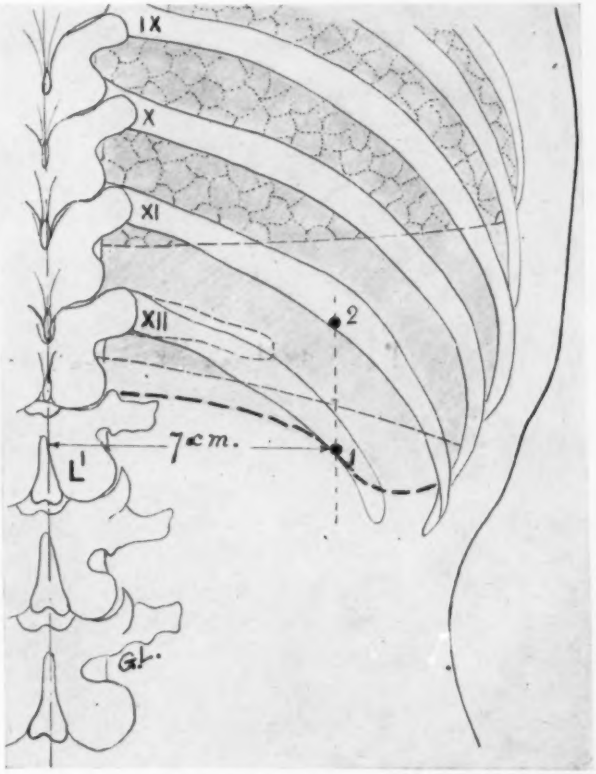

FIG. 13.—Relation of the site of puncture to the right costodiaphragmatic sinus. The needle passed through 1 is always below the pleural reflection. The heavy dotted lines show the level at which the pleura sometimes meets the lower border of the 12th rib. In a few cases the pleura follows the lower border of the rib 2 or 3 cm. further outward. Puncture through 2 is faulty technic.

Site of Injection.—Another important factor of success is the position of the point of the needle on the lateral aspect of the vertebral column. This can be accurately determined by tactile sense as much as by the pressure needed to inject the anæsthetic fluid. When the needle has been placed correctly, its point lies in the retroperitoneal tissue whose laxity offers very little resistance to the injected fluid and the syringe is discharged as easily as in the case of a perfect caudal injection. When the syringe is disconnected in order to be refilled a few drops of the injected solution very frequently flow out of the needle, and it occasionally happens that in puncturing the opposite side the needle brings out fluid from the injected side as soon as it

has reached the correct level. Kappis advises injection at a distance of ¼ to ½ cm. laterally to the vertebral column. This is hard to realize, especially when the most important recommendation is to keep constant contact with the body of the vertebra. Tactile sense is the only guide to prevent mishaps and carry the point of the needle approximately to the union of the anterior and lateral aspects of the vertebra. I am contented with pushing the needle 1 cm. further in, soon after its point has lost bony contact ventralward.

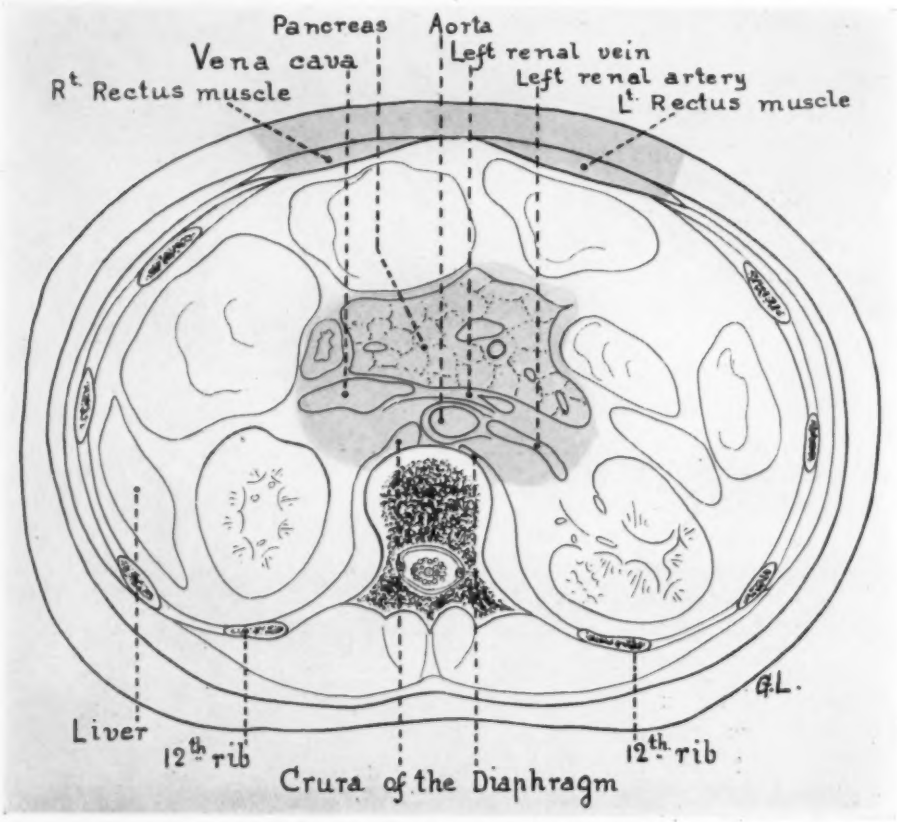

FIG. 14.—Diagrammatic illustration of the anæsthetized areas (gray) resulting from the association of abdominal field-block with splanchnic analgesia.

The needle must not scratch the periosteum or be forced through the margin of the intervertebral disk which is prominent and rather soft. It must pass freely alongside the body of the vertebra and be tangent to the bone including its tendinous and ligamentous coverings.

Extent of Analgesia.—The analgesia resulting from bilateral injections of the sympathetic plexuses of the upper abdomen (splanchnic nerves and solar plexus) involves only a relatively small portion of the posterior abdominal wall, especially the area under the direct influence of the operative manœuvres accomplished on the stomach, duodenum, pancreas and gall-bladder. The abdominal field-block which is always associated with the splanchnic in-

jections controls the anterior wall of the abdomen within the limits of the recti muscles. There exists between these posterior and anterior anæsthetized areas (Fig. 14) lateral zones of normal sensibility which must be treated with respect. Packing must, therefore, be light and gentle, avoiding the lateral walls. Traction on the organs must be gentle and gradual until the full limit of operative requirement is reached.

REPORT OF CASES

Case I.—C. B., male, age fifty-four years was admitted to the North Hudson Hospital, Weehawken, N. J., on October 31, 1923.

Chief Complaint.—Pain in the epigastrium for the past 12 years, intermittent and accompanied by vomiting. Hæmatemesis and melæna especially during the past 4 years. Loss of strength. Loss of weight (15 pounds in the last 3 years).

Physical Examination.—Color pale, nutrition poor, anemia. Teeth, many decayed. Lungs normal. Heart apparently slightly enlarged, sounds normal, no murmurs. Abdomen tender over the epigastrium and left hypochondrium, especially near the middle line; some rigidity, but no distention. Skin pale, yellowish. Temperature: 99°4. Pulse: 80. Respiration: 20. Urine examination: Negative.

Blood Examination.—On November 2nd, examination showed red blood cells 2,120,000 and hæmoglobin 20 per cent. The patient was transfused twice: November 5th (1500 c.c.) after which the red blood cells went up to 2,384,000 and hæmoglobin to 65 per cent.; and November 13th (1000 c.c.). Subsequent examinations made on November 14th and 30th showed red blood cells 3,600,000 and 4,112,000, hæmoglobin 60 per cent. and 62 per cent. respectively. Blood pressure varied between $\frac{110}{45}$ and $\frac{105}{60}$.

Provisional Diagnosis.—Gastric ulcer and possibly carcinoma of the stomach.

Operation.—On November 15, 1923, gastrectomy was performed under regional anæsthesia by Dr. Louis F. Lange of West Hoboken, N. J., assisted by Doctor Roberts and myself.

Palpation of the stomach revealed the presence of an ulcer near its lesser curvature, involving its posterior wall and adherent to the pancreas, practically about 2½ inches from the pylorus. Lesser and greater omenta ligated and stomach secured between two clamps applied parallel to the median plane of the body about 2 inches below the cardia. Stomach resected between the clamps. Distal segment turned over to the right and ulcer dissected leaving part attached to the pancreas. Duodenum sectioned and stump buried. Transmesocolic modified Polya gastrojejunostomy.

Operative and Post-operative Observations.—The operation lasted an hour and a half and was absolutely painless. The pulse was of good quality and remained practically unchanged throughout the operation. There was a short period of nausea while manipulating the stomach, accompanied by light sweats and pallor of the face; but there was no evidence of shock. Breathing was normal. The abdomen was completely relaxed. The post-operative course was excellent except for hiccough which lasted 3 days. There was no gastro-intestinal distention; no gas pain. The temperature was never higher than 99°4. Convalescence was easy and rapid. The patient left the hospital 18 days after the operation.

Case II.—W. F., male, age thirty-four years was admitted to the United Hospital, Portchester, N. Y., on November 29, 1923, for operation the next day.

Chief Complaint.—Vomiting (6 months' duration) practically everything for the past two months. Pain in the epigastrium 1 to 2 hours after meals. Loss of strength. Loss of weight from 135 pounds to 119 pounds. His best weight had been 150 pounds.

Past History.—Perfectly well up to the time he was "gassed" in the World War (August, 1918), stomach trouble began soon after.

Physical Examination.—Cheeks sunken, color pale, nutrition poor. Foul odor to

breath. Tongue moist and covered with thick brownish fur. Teeth, many decayed. Tonsils enlarged and cryptic. Lungs and heart apparently normal. Abdomen soft and flat. Visible and palpable mass about the size of a small orange just above and to the right of the umbilicus. Temperature: 99°4. Pulse: 80. Respiration: 20.

Urine Examination.—Negative.

Blood Examination.—Red blood cells 5,000,000; leucocytes: 7,600. (Lymphocytes 29 per cent., polymorphonuclears 71 per cent.); hæmoglobin: 60 per cent.

Provisional Diagnosis.—Ulcer or carcinoma of the pyloric end of the stomach.

Operation.—On November 30, 1923, gastrectomy was performed under regional anæsthesia by Dr. Charles W. Walker of Rye, N. Y., assisted by Doctor Quinlan of Portchester and myself.

Exposure of the stomach revealed the presence of a large callous ulcer which had perforated into the head of the pancreas and involved the pyloric end of the stomach and the duodenum. The common bile-duct was not involved. Dissection of the ulcer from the pancreas leaving its base which was 5 cm. in diameter. Dissection of the duodenum continued into its second portion. Section of duodenum at margin of ulcer. Closure of duodenal stump. Resection of nearly two-thirds of the stomach followed by trans-mesocolic gastrojejunostomy end-to-side.

Operative and Post-operative Observations.—The operation lasted almost 3 hours and was absolutely painless. The abdominal wall, however, was reinjected to allow closure.

The pulse showed no change in strength and frequency. Respiration was a little shallow, but there were no signs of distress at any time; no signs of shock. The first manipulations on the stomach induced a nauseated condition which subsided rapidly by deep breathing. The temperature remained practically normal the days following operation, the highest being 100°4 on the second day. Urine output was 22 ounces the day following the operation and continued at the rate of 20 ounces daily. There were no gas pains; the abdomen was flat and soft. The patient was able to sit up in bed the next day by his own means and was up five days after the operation. His convalescence was easy and rapid. He gained 2½ pounds in two weeks and left the hospital 19 days after the operation.

Preparation of Patients.—It is customary to lower the psychic element of the patient to a favorable level, in order to accomplish under the best conditions the various steps of the injection technic. Kappis[16] orders veronal 0.50 gm. (7½gr.) the evening before the operation and morphin 0.01 gm. (1/6 gr.) and scopolamin 0.0003 gm. (1/200 gr.) only half an hour before the operation is begun. Hoffmann[9] gives 1 gm. of veronal the evening before, and 1 to 1½ hours before the operation pantopon 0.02 gm. (⅓ gr.) and scopolamin 0.0002 - 0.0003 gm. (1/300 - 1/200 gr.). Buhre's patients usually receive scopolamin 0.0003 - 0.0005 gm. (1/200 - 1/120 gr.) and laudanon or narkoplin 0.03 gm. (½ gr.) or morphin 0.01 gm. (1/6 gr.). Nölle[34] injects ½ hour before the operation morphin 0.02 gm. (⅓ gr.) and scopolamin 0.0005 gm. (1/120 gr.). His patient is in twilight sleep during the operation and sleeps for several hours after. This dose is considered too high by those who are proficient in the art of inducing regional anæsthesia Veronal given the evening before is a good procedure, provided the operation is performed in the morning. I am in the habit of ordering a hypodermic injection of morphin 1/6 gr. and scopolamin 1/300 gr. given one hour before the patient is brought in the operating room; but if the patient is still aprehensive a similar dose is repeated immediately before the anæsthesia is begun. In the cases reported today, Doctor Lange's patient received morphin 1/6 gr.

only just before the anæsthesia was begun, a procedure which must not be encouraged, the injection being, as a rule, ineffectual. Doctor Walker's patient was prepared in the usual manner. We must emphasize the necessity of blunting consciousness in every case, but we make it our duty to caution against the use of heavy doses of narcotics which might be detrimental to the patient and defeat the aims of the regional method.

Induction of Anæsthesia.—Regional anæsthesia in both cases consisted in the injection of the splanchnic nerves on both sides by the posterior route of Kappis associated with field-block of the upper anterior abdominal wall. Only one injection was made on each side of the body of the first lumbar vertebra, due consideration being given to the coefficient of correction of the costovertebral angle discussed in this paper. The contents of two capsules (1 gm.) of pure Neocaine * were dissolved in 200 c.c. of sterile normal salt solution, thus making a 0.5 per cent. solution, and 15 drops of adrenalin chloride solution (1 : 1000) added to it. Of the 200 c.c. thus prepared 40 c.c. were injected on each side of the vertebral column at the level of the first lumbar vertebra and the remaining 120 c.c. used for the abdominal field-block. Laparotomy was begun almost immediately after the last injection in the abdominal wall.

FIG. 15.—Case I. Mr. C. B. (Doctor Lange's patient.) Before operation.

It is interesting to note that the 1 per cent. solution hitherto mostly recommended can be reduced to 0.5 per cent. without altering to any appreciable degree the quality of the analgesia required for operations on the upper abdomen; and since 200 c.c. is about the minimum amount of solution employed in these cases, the use of 0.5 solution for the splanchnic injections is more beneficial to the patient—especially if he belongs to the bad-risk group.

Pulse and Blood-pressure.—Kappis'[16] clinical observations are that "the pulse is generally accelerated, the blood-pressure sometimes sinking from 15

* Neocaine is a French product whose toxicity is very low. It is a powerful anæsthetic drug, which contains no adrenalin. It is sterile and readily soluble in normal saline solution. I have been using it for the last 6 years with very gratifying results.

to 20 mm. mercury sometimes unaffected and sometimes rising from 10 to 15 mm. mercury."

Hoffmann[9] says that generally the frequency of the pulse is somewhat reduced from 4 to 12 beats. In one case reported by Metge[27] there was an imperceptible pulse of short duration, associated with considerable fall of blood-pressure (125 to 25 mm. hæmoglobin). My experience is that the pulse rate generally increases during the induction of anæsthesia, then gradually goes back to normal or a little below normal; the frequency and strength of the pulse depending at the outset on the adrenalin contained in the solution and later on the condition of the blood-pressure.

FIG. 16.—Case I. Mr. C. B. (Doctor Lange's patient.) Three months after operation.

Hoffmann[9] found that if the induction of splanchnic analgesia preceded the abdominal field-block in some cases the blood-pressure fell from 3 to 20 mm. (Riva-Rocci), in others it remained unchanged. If the procedure were reversed the blood-pressure rose slightly. In 78 cases of the 104 cases reported by Metge[27] the blood-pressure dropped to below one-half of what it was before and after operation; but this did not seem to have great clinical significance. Schmidt[41] did not find any constant action of splanchnic anæsthesia on blood-pressure, and he believes that the psychic element seems to interfere with the results.

In most of my cases the blood-pressure was not taken during the operation, because the general aspect of the patient was satisfactory. But this does not mean that the blood-pressure was not influenced by the anæsthesia, for it must be remembered that in anæsthetizing the abdominal sympathetic system one must expect a fall in the blood-pressure due to loss of tonicity of the splanchnic vessels with a resulting flood of that territory at the expense of the peripheral circulation. Spinal anæsthesia is associated with similar disturbances of practically the same origin. These disturbances are, however, much greater in the case of spinal anæsthesia because of the extent of the territory involved in the anæsthesia needed for the upper abdomen.

After-effects.—The immediate after-effects associated with splanchnic

analgesia are chiefly those brought about by the unavoidable disturbances in the blood-pressure, *viz.*, pallor, cold sweats, nausea, vomiting, slow and feeble pulse. Their occurrence, however, is not extremely common and far from being alarming. According to Kappis[16] palor is more than when using other regional procedures, and is perhaps due to more rapid absorption of adrenalin or to reflexes resulting from anæsthesia of the splanchnic nerves or sympathetic system. My experience is that nausea is of frequent occurrence and ordinarily associated with pallor of the face. Vomiting is rare and is seldom disturbing to the surgeon, since the patient is asked to take a few deep breaths every now and then.

FIG. 17.—Case II. Mr. W. P. (Doctor Walker's patient.) Before operation.

Kutscha-Lissberg[21] in a critical review of his cases speaks of too rapid absorption of the anæsthetic drug with consequent intoxication, occasionally prompt collapse, and sometimes late conditions such as atony of the stomach and intestines. Similar reports have not yet come from men thoroughly familiar with the regional method and I personally have not made such observations. I see no reason why there should be prompt collapse following the injection of novocaine in the retroperitoneal tissue. Intravenous injections of the anæsthetic drug or heavy doses of adrenalin might have caused the collapse. Late disturbances in the tonus of the gastro-intestinal tract can hardly be attributed to splanchnic analgesia which rather increases peristalsis during its period of activity.

Apart from the symptoms incidental to the fall of blood-pressure, which once established does not create further disturbances, splanchnic analgesia is not associated with untoward effects worthy of consideration.

Post-operative Condition.—Post-operative and post-anæsthetic conditions are ordinarily good. Vomiting is rare. Shock is considerably minimized, if at all present. The after-effects are certainly less severe with splanchnic analgesia than with general narcosis in operations on the stomach and the gall-bladder, which are those for which it is preferred. It must be emphasized that splanchnic analgesia does not prevent pulmonary embolism nor can it

improve pathological conditions present at the time of the operation, as is generally inferred; but it certainly minimizes the risk of pulmonary complications and leaves undisturbed the central nervous system and the liver, which are the organs on which life chiefly depends. In 112 operations under splanchnic analgesia Metge[27] had 3 fatal pneumonias; but he had 7 similar fatalities in an equal number of operations performed at the same time under general narcosis. Most writers are agreed that a good splanchnic analgesia affords better operative facilities than general narcosis and does not interfere with the post-operative course of the disease.

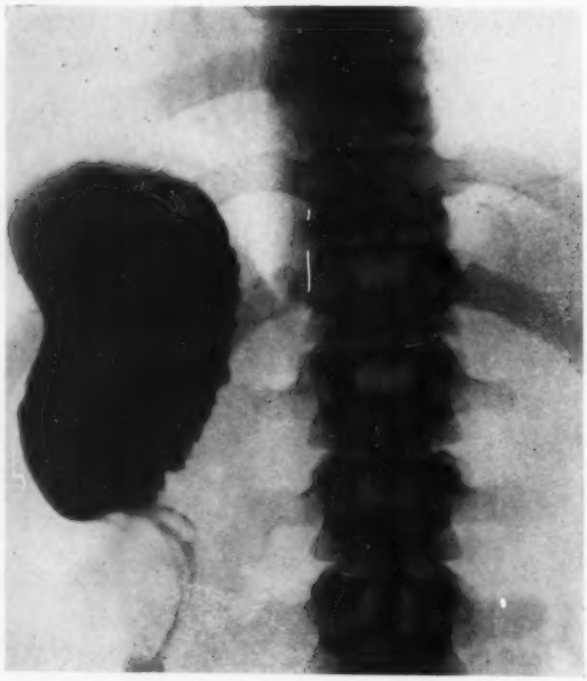

FIG. 18.—Case II. Mr. W. P. (Doctor Walker's patient.) Two months after operation.

CONCLUSIONS

Splanchnic analgesia is the most recent form of regional anæsthesia applicable to abdominal surgery, especially that of the upper abdomen. It has in the last five years, attracted the attention of many surgeons who have used it in a fairly great number of cases with very gratifying results.

Of the three original methods Wendling's is not recommended, Braun's has a few partisans, Kappis' enjoys the favors of the majority of writers and certainly has the greatest practical value.

Modifications of Kappis' technic are worthy of interest, because they caution against pitfalls and contribute to greater success.

Best results are obtained when the solution is deposited between the semilunar ganglia and the renal pedicles.

Variations exist in the inclination of the 12th rib on the vertebral column which are responsible for a certain number of failures. These failures can be reduced to a minimum by applying the coefficient of correction of the costo-vertebral angle, that is puncturing on the lower border of the 12th rib opposite the upper extremity of the first lumbar spinous process.

It is perfectly safe to approach the splanchnic area from the back. By keeping close contact with the vertebral column and following the technic described for that route it is impossible to make an intraspinal or an intravenous injection.

The concentration of the solution can be advantageously reduced from 1 per cent. to 0.5 per cent.

Splanchnic analgesia has a very great field. There seems to be no contra-indication to its use. It needs only be applied judiciously by those already familiar with the regional method.

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SUBLINGUAL FIBROMA

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It is so rare to observe a chronic swelling involving the sublingual space that the diagnostic possibilities do not readily come to mind. The subjoined case therefore was of interest both for diagnosis and treatment. No similar type of growth has been found in the literature, although cases may have been overlooked, the difficulty of proper indexing being considered.

Case.—Mr. C. R., aged thirty-nine years, was admitted to the New Haven Hospital, November 29, 1921, referred by Dr. F. N. Sperry of New Haven, Conn. He complained of a swelling under the tongue. For two and one-half years there has been an increasing difficulty in swallowing and in speech and an enlarging growth under the tongue and below the lower jaw. The condition has made a slow but steady progress. He has not had any visual disturbance, nervousness, irritability, tremor, sweating, palpitation or rapidity of the heart.

He had had measles, scarlet fever and typhoid fever as a child and inflammatory rheumatism seven years ago (1914) for nine weeks during which time he was in a hospital at Bridgeport. Otherwise his past history was unimportant.

The physical examination was normal throughout except for the local condition for which he entered. The tongue was large, moist and slightly coated; it was pushed upward, in its posterior portion almost meeting the hard palate. The swelling was slightly more prominent on the right than on the left side. The mass showed no signs of inflammation; it was firm, had a tense elastic feel, and seemed to be deep under the substance of the tongue itself. Nothing definite could be made out under the tongue anteriorly; it was not possible to examine the tonsils or pharynx because of the obstruction to the view. Externally there was a smooth enlargement of both submaxillary regions and a decided fulness in the submental area. Palpation gave the impression that there were several glands in each of these positions but they did not seem to have a connection with the deeper underlying mass. Bimanual palpation with one finger on the tongue and the other hand externally in any of the three triangles indicated that the swellings were continuous. The voice had a nasal quality, the patient talking as if his mouth were full. The thyroid gland and the isthmus could be palpated in the normal position.

The temperature, pulse and respirations were within normal limits. The red blood count was 5,200,000; Hæmoglobin 95 per cent. (T.). There were no abnormalities of the red cells. Blood grouping II Jansky. The white blood count was nine thousand and eight hundred with 72 per cent. polymorphonuclears. Eosinophiles, 4 per cent. Basophiles 1 per cent. Lymphocytes 18 per cent. Mononuclears 5 per cent. The urine examination was normal. The blood Wassermann test was negative.

In summary, we had a man of thirty-nine years, who complained of a slowly increasing difficulty in swallowing and speech. The condition had been present for at least two and one-half years; and on examination a smooth, non-tender, semi-elastic feeling

mass was present in the posterior sublingual region, pressing the base of the tongue upward against the palate, and bulging the submaxillary and submental triangles externally.

In an attempt to arrive at a diagnosis the following conditions were considered:

(1) *Cystic Conditions.*—Ranula—against this diagnosis was the freedom from involvement of the tissues under the tongue anteriorly—the position most frequently occupied by these cysts; the solid consistency of the tumor mass, although a tense cyst might feel similarly; and the failure of the growth to transmit light when attempts were made at transillumination. *Thyro-glossal cyst.*—No evidence of persistent sinus at the foramen cæcum (very difficult to examine) or exteriorly anywhere in the

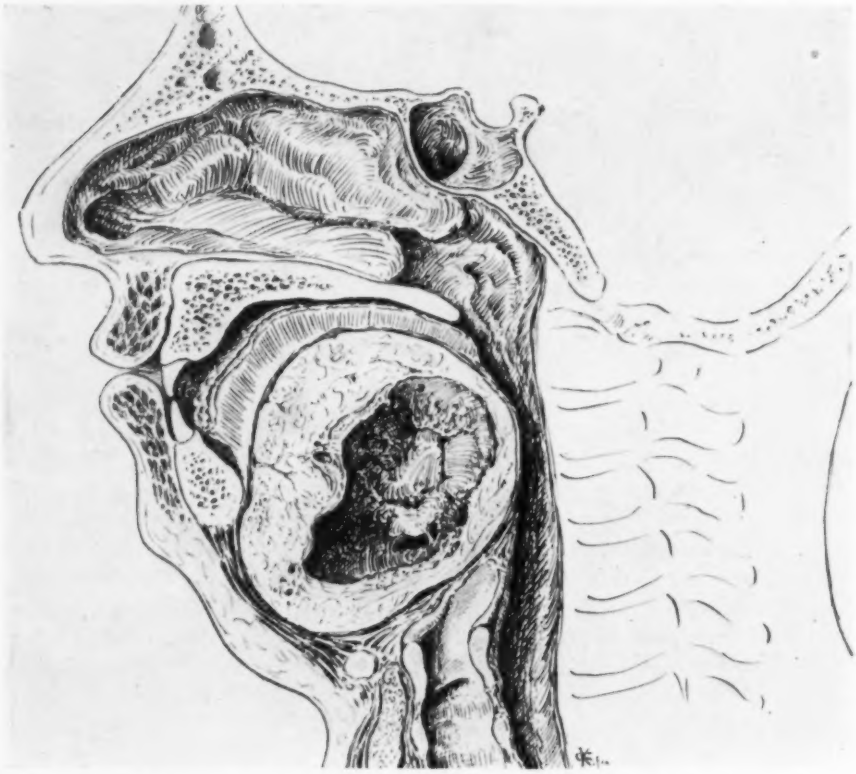

FIG. 1.—Cross-section of drawing (scale) showing the tumor in the position it occupied.

neck; and the same objections as to ranula, although the situation of the growth was not against a thyro-glossal origin. Dermoid cyst could not be ruled out.

(2) *Solid Tumors.*—Lipomas, fibromas, endotheliomas and mixed tumors have all been described as arising in the tongue itself and although reports of such tumors occupying the posterior sublingual region have not been found in the literature, there is no reason to believe that the growth might not take this direction. A case of sarcoma of the tongue "which sprang from the base of the tongue and grew downward and forward, causing a bulging beneath the chin", was described by Schulten[1]. A lingual thyroid was also taken into consideration. Such a growth could occupy the exact situation of the tumor under discussion. Almost always these goitres are apparent in examining the posterior portion of the tongue itself. The lack of thyroid symptoms and the fact that the gland could be palpated in the normal position were against such a diagnosis.

It did not seem possible to make the diagnosis without operation but the patient refused any radical procedure, although finally reluctantly consenting to the removal of a small bit of the growth under procaine anæsthesia.

November 30, 1921.—Operation. Exploration and removal of specimen for diagnosis. Under procaine a transverse incision one inch long was made over the most prominent portion of the swelling in the right neck. This incision was placed low in order to avoid the submaxillary gland. The tissue removed appeared solid in consistency and bluish white in color. The nervousness and apprehension of the patient

FIG. 2.—Lateral view of the tumor. The scale is in inches.

made a more thorough examination out of the question. The specimen on pathological examination was reported to be a flattened out and compressed salivary gland.

Between November 30, 1921 and October 14, 1922, the growth was subjected to seven radium treatments but without noticeable result. He finally consented to operative removal of the growth.

October 14, 1922.—He was taken to the operating room and gas-oxygen anæsthesia given. During the course of the administration the patient became very cyanotic and stopped breathing. It was necessary to pull forward the tongue and to use artificial respiration. Finally just as a skin incision had been made for tracheotomy the patient took a breath. During this performance the operator had his finger over the base of the tongue pulling it forward in order to get a clear air-way and in this way

it was possible to palpate a large boggy tumor. It seemed to occupy the whole area of the tongue and anterior portion of the pharynx. The tumor felt as though it were full of fluid. On account of the difficulty in the anæsthesia the patient was returned to the ward, future operation to be decided on after a preliminary tracheotomy.

November 18, 1922.—Under procaine anæsthesia a preliminary tracheotomy was done low in the neck just above the sternal notch. The patient had considerable irritation, coughing, and a feeling of suffocation when the tube was introduced, but after 5 or 10 minutes he calmed down and stood the operation well. He returned to the ward in good condition. The tube was tolerated well and the patient breathed very comfortably.

November 21, 1922.—The second stage of the operation was undertaken. After a very difficult beginning, the patient was finally anæsthetized through the tracheotomy

FIG. 3.—Cross-section showing the degenerated cyst-like cavity.

tube, it being necessary to pack the naso-pharynx and also to introduce a mouth gag in order to prevent some breathing by the natural air-way. Operation was started with a transverse incision mid-way between the point of the mandible and the hyoid bone extending from the sternocleidomastoid anterior border to a similar position on the opposite side. The platysma was divided, the anterior belly of the digastric across its mid-portion, the mylohyoid and the geniohyoid and a portion of the geniohyoglossus. At the bottom of the incision, the anterior surface of the tumor, light pinkish in color came into view. A thin areolar tissue about the growth was divided and the mass separated from the surrounding tissue by gentle finger dissection. It occupied a position which pushed the posterior part of the tongue up against the hard palate. It also extended backward so that in getting around it, it was possible to feel the anterior border of the vertebræ (Fig. 1.) The growth was worked free all around and delivered intact with considerable difficulty through the small incision below the chin. There was very little bleeding. It was not necessary to tie off either the lingual or facial vessels, and the hypoglossal nerves on each side were retracted from the field of operation. A large dead space left by the removal of the growth was drained with two Penrose drains and suture was made with fine plain catgut approximating the divided muscles as accurately as possible. The skin incision was closed with silk.

During the operation the upper pole of the left thyroid gland came into view and appeared to be normal. The patient stood the operation well and returned to the ward in good condition. The post-operative convalescence was without special incident.

December 2, 1922.—He was discharged. General condition excellent. Vital signs normal. Tracheotomy wound completely healed. Operative incision healed except where the drains were inserted. There had been no infection. No paralyses. Breathing improved and it was decidedly easier to swallow.

February 11, 1923.—Patient wrote a letter today saying he had gained thirty pounds since discharge from the hospital and was feeling fine.

Pathological Examination.—Grossly the tumor was a light purplish pink in color with occasional yellowish patches. It was slightly nodular, firm and surrounded by a thin veil of reddish areolar tissue. It measured 7.5 cm. by 7.0 cm. by 5.0 cm. in diameter (Fig. 2) and weighed 170 grams. It was cut open in its long axis, revealing a cystic area in the centre. This area measured 5.0 cm. by 4.0 cm. by 3.0 cm. in diameter. The walls of the cystic cavity were covered for the most part by a thin blood clot, reddish brown in color. There was also a considerable amount of yellow serous fluid. The mass of the tumor around the cyst was composed of solid pink tissue arranged in irregular clumps of interlacing fibrous appearance (Fig. 3).

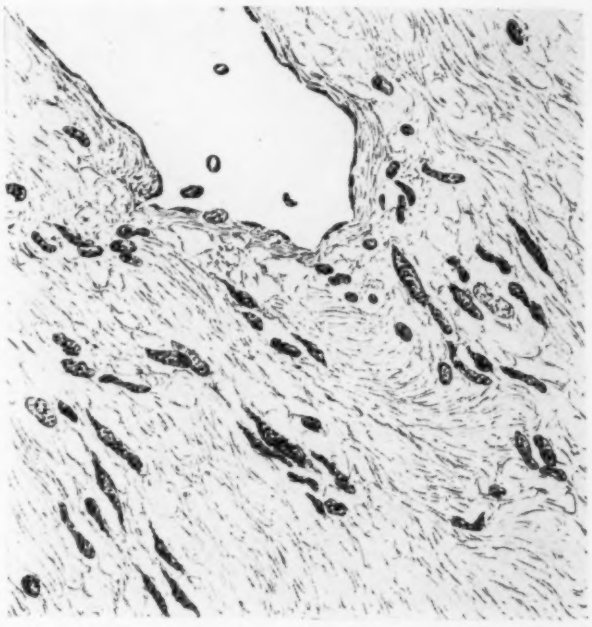

FIG. 4.—Drawing of microscopic section. The fibroblasts and fibrous intracellular tissue is well brought out. In the upper left hand corner is a large vascular space.

Microscopic examination showed a practically uniform picture throughout the five blocks examined. The sections consisted of elongated, faintly blue-staining nuclei interspersed with a large amount of pink fibrillar substance. There were occasional large endothelial-lined blood spaces. No true cyst wall was made out (Fig. 4). Special stains were made for muscle and nerve but no such fibrils could be demonstrated. The sections were examined by several prominent pathologists who all agreed on the diagnosis of fibroma.

It is interesting to speculate on the origin of such a growth. Owing to the fact that the aberrant thyroid is often seen in such a situation, it is possible that this tumor had its origin in the fibrous tissue remains of the thyroglossal migration tract. There are, however, so many other layers of connective tissue from which the tumor could have grown that no definite conclusion can be reached. The cystic area in the tumor may well have been a radium effect. There was no cyst wall so that we must assume that the broken down tissue was the result of necrosis caused either by the radiation or a failing blood supply. The position of the area in the tumor corresponded

exactly to the point where the maximum cross-fire radiation effect would occur; and it is also to be noted that the areas which would receive least raying on account of their position under the mandible and tongue, showed the most solid portions of the tumor.

Summary.—A chronic swelling involving the posterior sublingual region in a middle-aged man, was treated for about one year by radiotherapy without apparent effect. The growth was then removed through a submental incision and microscopic examination showed it to be a fibroma. No similar case has been found in the literature.

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ACUTE PANCREATITIS*

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Affections of the pancreas due to trauma, either external on the abdominal wall, or internal, produced at operation, are not to be considered here, neither are the acute affections occurring in the course of a general pyæmic infection. The condition I am concerned with is the acute affection of the pancreas, appearing suddenly and incapacitating the patient in a short time, frequently leading to death. Text-books in general make a distinction between acute hemorrhagic pancreatitis, apoplexy of the gland, suppurative pancreatitis, and necrosis of the gland. Clinical observation however seems to point to the conclusion that these different varieties are really the same condition in various stages of development, or presenting different degrees of severity.

Owing to the many contributions to this subject, both by clinicians and by laboratory workers, the disease has become fairly well known. We may safely say that we know acute pancreatitis clinically, we know the associated conditions, but we do not definitely know its etiology. There are two viewpoints regarding the cause of the disease:

1. That it is due to the entrance of bile or duodenal contents into the pancreatic duct and that the symptoms are due to infection or to ferment action.

2. That it is an infection carried to the pancreas by means of the lymphatics.

Speaking of acute pancreatitis, of duct-borne infection, or of infection carried by the lymphatics all presuppose that we are dealing with a bacterial invasion. A study of the literature, however, and my personal observations lead me to doubt that this assumption is correct. For this reason I desire to present the histories of six rather carefully observed patients, which seem to support the theory that infection as such has nothing or little to do with acute pancreatitis, but that it is due to the action of liberated pancreatic ferments on the surrounding tissue.

Case I.—F. H. A man thirty-nine years old, admitted to the Lenox Hill Hospital, forty-six hours after the onset of an acute abdominal condition. Two days previously he had enjoyed a good dinner and was feeling fine when he was suddenly seized with severe colicky pains in the epigastrium, accompanied by vomiting. His physician made a diagnosis of cholelithiasis and administered morphin which temporarily relieved the pain but did not control the vomiting. His condition grew worse, he went into collapse, and a diagnosis of perforation of the gall-bladder was made and the patient sent to the hospital. On admission he was found to be gasping for air. His skin was blue and cold and covered with perspiration. The pupils were dilated to maximum. There was constant vomiting of yellowish non-odorous fluid. His pulse was not perceptible

* Read before the Surgical Section of the New York Academy of Medicine, October 5, 1923.

and the heart action was weak, and rapid, 156 per minute. The chief indication was attention to his collapsed condition. All known measures were resorted to and the patient recovered sufficiently to be able to give a description of his illness. He stated that he had a severe attack of abdominal pain two years previously which had been diagnosed as cholelithiasis, but which did not resemble his present attack. Since that time he had had several minor attacks of pain, never severe enough to consult a physician. He led a regular life and took alcohol in moderation. Physical examination further showed that the heart and lungs were not diseased. The abdomen was moderately distended, but there was no board-like rigidity such as one finds in peritonitis. There was, however, exquisite tenderness over the upper abdomen, most marked just above the umbilicus. The thick walls of his abdomen made examination difficult, but no free fluid could be made out. No organs or abnormal masses were palpable and the area of liver dulness was not diminished. Temperature was 102.6 per rectum. Respiration 58. The vomitus contained blood on chemical examination, nothing else abnormal. Fæces were negative; they were partly formed, had normal color, normal odor and did not contain blood. Urine could not be obtained, but an examination made by his family physician before his admission had shown neither albumen, sugar nor bile.

A diagnosis of acute pancreatitis was made, based on the three principal symptoms, sudden onset of colicky pain in the epigastrium, constant vomiting and collapse, and the absence of symptoms or signs pointing to some other disease. Under the existing conditions an operation could not be done and the patient died two hours after admission, 48 hours after the onset of his illness.

An autopsy was done within a few hours after death and the abdominal examination showed a large amount of light brown turbid fluid without odor. The peritoneum was slightly reddened but smooth. Fat necroses were found scattered over the entire peritoneum, especially in the omentum, the mesentery, and in the vicinity of the pancreas. Most of them were small whitish opaque spots, others were as large as a lentil, and in the fatty tissue around the pancreas large strips and plates had formed. The pancreas was considerably enlarged, surrounded by a mass of infiltrated fat, together with which it weighed 650 gms. The liver was about normal size and appearance, the gall-bladder was enlarged, its walls were thick and it contained 65 gall-stones of various sizes. The excretory ducts were patent.

The pancreas was split in its long axis and examined. It showed tissue which in parts looked reddish, in other parts fairly normal. Through its entire length passed a strip of soft, almost black necrotic tissue, which was wider in some places than in others. In addition to this long strip of necrotic tissue there were smaller foci scattered through the gland. After much difficulty the excretory duct was found, and the following interesting observation made. The duct of Wirsung in its entire extent passed through the middle of the necrotic strip of pancreas. About 3 cm. of its lower end were dilated and almost entirely destroyed, in parts the wall could not be made out so that it was impossible to find the entrance into the papilla of Vater. All the pancreatic tissue surrounding this lower portion of the duct was entirely necrotic. Following the duct upwards into the body and tail of the gland showed it to be better preserved, but the pancreas surrounding it was likewise necrotic. The exact relation of the duct of Wirsung to the common bile duct could not be made out owing to the extensive necrosis. Both ducts were patent and did not contain calculi. Microscopical examination of the pancreas showed a varied picture. Some areas were entirely necrotic, others less so and still others fairly well preserved. In a general way the destructive process extended from the larger ducts and from the interlobular connective tissue. Many lobules were well preserved, especially in the centre, while the acini of the periphery were more or less destroyed. In other places entire lobules had been destroyed, so that no acini could be identified, only irregularly arranged cells which were stained poorly. Into some of these necrotic foci hemorrhage had taken place or a granular pigment had been deposited. Many of the necrotic foci were enclosed by a dense

inflammatory zone. This could be noticed especially in the fatty capsule surrounding the pancreas. But wherever the necrotic process gradually passed over into more normal tissue, no inflammatory zone was present.

In addition to the pancreas the other organs of the body were affected, probably the result of the general toxæmia. The liver showed subacute hepatitis, many of the cells were in a state of well advanced fatty degeneration. Heart and kidney showed parenchymatous degeneration.

CASE II.—Mrs. E. L., fifty-two years old, was admitted August, 1916, complaining of excruciating epigastric pain and vomiting. The symptoms had started suddenly on the previous day. She had had eight similar attacks. Her abdomen was slightly distended. The diagnosis rested between perforated gastric ulcer and pancreatitis. She was extremely sick and suffered terribly.

Immediate abdominal section was done. Median incision. Large amount of yellowish turbid fluid. Fat necroses scattered throughout the fatty tissue of the omentum and especially about the pancreas. Stomach normal. Gall-bladder somewhat thickened, not acutely inflamed, filled with stones. Pancreas enlarged and hard. Peritoneum and fat covering it were bluntly separated and three gauze tampons inserted. Profuse drainage continued. No excoriation of the skin. After about three weeks a large necrotic slough was extruded which on examination showed enough structure to indentify it as pancreas.

The discharge from the wound was examined for ferments, but none were found. A sugar tolerance test proved normal. After discharge of the necrotic pancreas, the wound secretion rapidly diminished in amount and the fistula closed. The subsequent history of this patient has been quite stormy. After operation she did fairly well, she was able to eat everything, but at times she had pain in the upper abdomen radiating to the back. In the fall of 1920, four years after her pancreatitis, I saw the patient during an attack of acute cholecystitis with jaundice. A cholecystectomy was done. No free fluid and no fat necroses were found. The head of the pancreas was palpated and found to be rather hard. The body and tail could not be definitely made out. Convalescence was uneventful.

In February, 1922, she again came under observation with symptoms suggestive of pancreatitis. She had had attacks of soreness in the upper abdomen, nausea and vomiting for 6 months and one of these attacks started a week ago. The night before I saw her, she had suddenly become much worse. Excruciating pain had developed in the epigastrium, accompanied by frequent vomiting, as often as every 15 or 20 minutes. She had become jaundiced in the course of the week. After several days observation I operated with the provisional diagnosis of obstructive jaundice, due either to a stone or adhesions. Operation was difficult on account of extensive adhesions. No free fluid or fat necroses. A penetrating duodenal ulcer was found on the antero-inferior border, about one inch below the pylorus. It was closed with a purse-string suture and a posterior gastro-enterostomy was then done. Examination at this time revealed that the abdomen below the transverse mesocolon was entirely free of adhesions and that the head of the pancreas was intact, while the body and tail were apparently missing.

In October, 1922, the patient was again admitted for symptoms similar to those of which she had formerly complained. She had developed a large ventral hernia through the median scar. X-rays for stone in the common duct or pancreas were negative. The function of the gastro-enterostomy was normal. She was again operated on, an exploration was done and the ventral-hernia was repaired. Since then she has been perfectly well, and today she enjoys a normal life.

CASE III.—Mrs. A. L., thirty-five years old, was admitted on August 25, 1921. She had been sick four days. The onset had been sudden, awakening her at 12.30 A. M. with pain in the upper abdomen so excruciating that she had been unable to speak. The pain radiated to the back. The previous evening she had eaten a heavy supper

and enjoyed it. A hypodermic of morphin brought temporary relief, and then the pain returned. The next morning she began to vomit and continued at short intervals all day, when it stopped after a morphin injection. Since that time she had not been free from pain but vomiting had not recurred. The patient gave a history of recurring attacks of pain in the upper abdomen since her last child was born one and a half years ago.

On admission her temperature was 99.8, pulse 98, respiration 22. She was a very obese woman which made examination difficult. She was slightly jaundiced, but did not look very sick. She still complained of severe pain in the epigastrium, to the right and particularly to the left of the median line. The abdomen was somewhat distended. There was no rigidity and no free fluid could be made out. There was marked tenderness over the entire upper abdomen, most marked over the gall-bladder region. A blood-count was not done. The urine contained a faint trace of albumen, but was otherwise negative. She impressed as a typical gall-bladder case and was operated on with that diagnosis.

A right rectus incision was made. A large amount of clear yellow fluid was present. As soon as the gall-bladder region was exposed fat necroses were noted in the fat surrounding it, as well as in the omentum and the gastro-hepatic omentum. They were scattered all over, but were most numerous about the pylorus and in the tissue overlying the pancreas. The gall-bladder was about normal size, did not look acutely inflamed but was tense and thick-walled. Bile could be pressed out. Stones were not felt. The duodenum was mobilized for exploration of the bile duct. No stones felt and no distention noted, although its lower end was surrounded by hard nodular pancreatic tissue. Attention now directed to the pancreas which was enlarged and hard. It was approached through the gastro-colic omentum. No gangrene or areas of softening noted. Three gauze tampons were inserted to the prepancreatic area and the abdomen was then partly closed.

On the second night the temperature went to 102.4, but by the fifth day it reached normal. The post-operative course was without incident. The drains were removed on the tenth day. There was at first profuse serous secretion containing small shreds of necrotic tissue. The secretion had no irritative action on the skin. Except for the presence of albumen for a few days after operation the urine remained normal. She was discharged healed on the thirty-fifth day. Since that time the patient has remained well except that she is troubled with hyperacidity. She is pregnant at present about four months, but this apparently has not produced any abnormal symptoms.

Case IV.—Mrs. L. C., thirty-eight years old, admitted September 13, 1921. Sudden onset day before with excruciating pain in the upper abdomen. It had been continuous ever since and so severe that she had been unable to rest. Shortly after onset she began to vomit, and this also had continued steadily. Her bowels had moved, and there were no urinary symptoms.

One year ago she had a similar attack, but not so severe. It responded to medical treatment. Since then she has had several minor attacks. She thinks they came on following an abortion. She has never been seriously ill. Her first child was delivered by Cæsarean section ten years ago. After that she had three children born normally, but the fifth was again delivered by Cæsarean.

On admission she was in extreme pain, tossing about in bed. Her skin and sclera had a yellow tinge. Her tongue was coated and the throat congested. She vomited frequently, just clear fluid, and belched considerably. The pain was situated in the epigastrium, spreading to both sides with equal intensity. The abdomen was not distended. There was no hernia through the scar. No peristalsis was noted. Palpation revealed slight rigidity over the entire upper abdomen, with very marked tenderness, somewhat more acute on the left side, but passing around both sides to the back. The temperature was normal, the pulse 70.

On the basis of the sudden onset with continuous severe epigastric pain, almost

constant vomiting, slight rigidity with very marked tenderness of the upper abdomen and the absence of inflammatory signs, a diagnosis of acute pancreatitis was made. Operation was advised but refused, and because the symptoms were not grave, we did not urge, especially as we were interested to know whether spontaneous cure would result.

On the following day the pain was somewhat less, most intense in the left upper abdomen. The jaundice had deepened somewhat, bile was present in the urine and the bile color index of the blood was 7 as compared with 2–4 normal. The pulse was 60, temperature 99.8, white blood-cells 16,800, polymorphonuclears 94 per cent. Tenderness and slight rigidity were unchanged and had extended to the lower abdomen. Vomiting continued. A duodenal tube was introduced but promptly vomited. On September 15, the fourth day after her disease began, the temperature gradually rose to 102.2, pulse 106, respiration 24. Pain became more severe. Slight rigidity and very marked tenderness, especially to the left of the median line, persisted. The white blood-cells were 16,400, polymorphonuclears 92 per cent. The diagnosis of acute pancreatitis was maintained, operation was urged and was accepted.

A median epigastric incision was made. A large amount of hemorrhagic fluid was found. Fat necroses were scattered over the abdomen; they were most numerous in the transverse mesocolon, about the duodenum and over the pancreas. An incision was made in the transverse mesocolon and the pancreas exposed. It was large and hard and surrounded by fat necroses and gangrenous looking tissue, a portion of which was removed for microscopical study. The entire head of the pancreas seemed involved. Considerable bleeding was encountered, due to the sloughing nature of the tissue and there was much difficulty in controlling it. The peri-pancreatic tissue was bluntly entered in several places and three gauze tampons were then inserted. Cultures were made from the peritoneal and retroperitoneal exudate. The gall-bladder was then opened. It was not acutely inflamed, but its walls were thickened. Several stones were removed and cholecystostomy then done. Her convalescence was a stormy one. On the third day she developed an unexplained œdma of the face which closed her eyes and lasted several days. The tampons were removed on the thirteenth day. The discharge was profuse and at times contained necrotic fragments of tissue and on the fifteenth day also considerable blood. It never produced excoriation of the skin. Laboratory findings were: 1. Wassermann negative. 2. Examination for pancreatic ferments in the discharge and fæces were negative. 3. The urine contained albumen for several days, bile only on the first day, and never sugar. 4. Cultures of the peritoneal and retroperitoneal exudate were sterile. 5. Cultures taken from the gall-bladder secretion on the ninth day contained colon bacilli. 6. Examination of the necrotic peri-pancreatic tissue removed at operation showed necrotic and suppurative fat tissue with hemorrhage. 7. Necrotic tissue discharged post-operative showed degenerated tissue composed of indistinct cells, and arranged in small groups, suggesting acini, probably necrotic pancreatic tissue.

Case V.—Mrs. A. C., twenty-five years old, was admitted January 27, 1922, complaining of pain in the upper abdomen which had started three weeks ago. She had had similar attacks at irregular intervals during the last two and a half years, the first one coming on a week after her last confinement. Each attack had started in the right upper quadrant, had then spread over the entire upper abdomen, and had been accompanied by frequent vomiting. Her present attack was the most severe. It had started in the middle of the night with excruciating pain and vomiting which continued uninterruptedly. She was seen by me in consultation on the third evening after the onset, and seemed to be suffering intensely. She was a big adipose woman, rather apathetic, and answered questions reluctantly. She looked pale, her eyes were sunken and underlined by deep rings. Pulse poor quality, 72. The temperature was subnormal. She seemed to be in shock. The abdomen was collapsed and soft all over. No rigidity at all. No organs palpable. Over the entire upper abdomen there was marked superficial and deep tenderness.

Removal to hospital was advised, but was refused by the patient, and she was lost sight of until she was admitted almost three weeks later. She stated that in the meantime her pain had gradually shifted from the right upper to the left upper abdomen, where it had remained as a steady dull pain, radiating to the back at the same level. For the first two weeks she had vomited whenever she took food, but less often during the last week. Her sleep had been disturbed by a great deal of pain, and she had lost fifteen pounds in weight. There had been no jaundice. Her bowels had been regular, and no blood had been noticed in the fæces. The temperature was 98.6, pulse 92, respiration 20. The physical signs had changed a great deal since the first examination. The abdomen was soft and relaxed, there was no superficial tenderness or rigidity. On deep pressure there was tenderness across the entire upper abdomen, most marked in the epigastrium and to the right of it. Corresponding to this tenderness a mass could be palpated which reached from the ensiform to the umbilicus and was nearer the surface in the median line than on either side. Its lower border was sharp and corresponded in general to the outline of an enlarged liver. It could be followed to the left as far as the costal margin. There was no tenderness or swelling in either costo-vertebral angle. No free fluid could be made out.

The patient was operated on the following day. Epigastric incision just to the right of the median line. No free fluid. Numerous fat necroses were found scattered throughout the omentum and in the fat surrounding the pancreas. The liver was small and soft. The gall-bladder showed no adhesions, its walls were thickened and it was filled with small stones. The posterior wall of the stomach was adherent to a large cystic mass occupying the region of the pancreas. The lower border of this mass was hard and nodular, and was formed of gastrocolic omentum which had been drawn backward in order to wall off the mass. It was this rather sharp border which had been mistaken for liver edge prior to operation. When lifting up the transverse colon a thickened nodular mass could be seen and felt surrounding the exit of the duodenum. Fat necroses were found scattered throughout the tissue.

It seemed then that we were dealing with a pancreatitis which had quieted down and which had formed a pseudo cyst retroperitoneally. A cholecystectomy was done first, beginning the dissection at the cystic duct. The pancreatic region was now entered by bluntly going in between the stomach and the transverse colon. The peritoneum was found to be much thickened. A large cavity, situated behind the stomach, was entered, and at least a pint of turbid fluid containing flocculi evacuated. A rough mass could be felt in the cavity, which after extraction proved to be a slough of the pancreas, perhaps about half the gland. A cigarette drain and one rubber tube were inserted into the cavity and another cigarette drain to the gall-bladder region, and the wound was then closed to the drain. The convalescence was uneventful. On the day after operation the temperature rose to 102, but reached normal by the sixth day and remained that way. Drainage was profuse for the first few days, at no time, however, excoriating the skin. On the ninth day the gall-bladder drainage and all sutures were removed, and on the fifteenth day also the drainage tube from the cyst. The patient was discharged completely healed after six weeks.

Laboratory Findings.—Urine negative for sugar all the time. Albumen present after operation, then disappeared. Blood count on admission: White blood-cells 9400, polymorphonclears 73, hæmoglobin 60 per cent. Wassermann negative. Pancreatic cyst-fluid negative for pancreatic ferments. Cultures from the peritoneal fluid, the pancreatic cyst fluid and gall-bladder were all negative.

Pathological Examination.—Inflamed gall-bladder, containing many stones. No bacteria in wall. Pancreatic slough so necrotic that structure could not be recognized.

Case VI.—Mrs. C. F. was admitted August 27, 1923, complaining of abdominal pain and vomiting. It had started two days ago with excruciating pain in the epigastrium which within fifteen minutes had spread diffusely throughout the entire abdomen. Soon after she developed pain in the back, just below the costal margin. From here the pain

radiated to both shoulders. It came on in frequently recurring spasms, did not seem to radiate from the epigastrium to the back, but in each area increased in severity simultaneously. Soon after the onset of pain she began to vomit at frequent intervals, and the night before admission she had vomited every fifteen minutes. The vomitus was greenish-yellow fluid without disagreeable odor. The bowels had moved and the stools had been normal.

The patient stated she had a similar attack two years ago, which lasted about two hours and was less severe, though accompanied by chills, fever, backache and vomiting. She had also had a slight attack nine years ago following a dilatation and curettage. Aside from the above attacks she had always been well. Her menstruation was normal The clinical picture was that of a patient in acute distress. She was unable to keep still, was tossing in bed, moaning with pain, and vomiting every little while. She begged that something be done to relieve her. The abdomen was slightly distended. There was no rigidity, and no free fluid could be made out. There was very marked tenderness, even on light pressure, over the entire upper abdomen, and extending downward on the right side, but most marked in the epigastrium. No organs could be made out. There was no icterus. Vaginal examination elicited much tenderness, but there was nothing abnormal to be made out. Her temperature was 99.4, pulse 96. The urine showed a heavy trace of albumen. No sugar. Blood examination showed white blood-cells 18,800, polymorphonuclears 88 per cent. A diagnosis of acute pancreatitis was made and operation was performed within a few hours after admission. A four-inch incision was made through the upper right rectus. Only a little fluid and scattered fat necroses were seen at first. The abdomen below the tranverse mesocolon was free from fluid and showed no other involvement. It was therefore protected with pads, and attention directed to the upper abdomen. A hemorrhagic exudate surrounded the liver and stomach, and infiltrated these organs. There was also a large amount of bloody fluid behind the peritoneum, surrounding the pancreas and duodenum, extending downward on the right side behind the peritoneum of the ascending colon. There was extensive necrosis of the tissue surrounding the pancreas. The tranverse mesocolon had been drawn backward and was adherent over this necrotic mass. The walls of the stomach were red and injected and intensely œdematous. The gall-bladder was normal in appearance, thin-walled, but filled with small stones. There were no adhesions. It was quickly removed, and the tissue overlying the pancreas was then bluntly entered, evacuating considerable sanguineous exudate. Three gauze tampons were placed into the peri-pancreatic tissue and a cigarette drain to the gall-bladder stump. Abdomen then closed in layers. At the end of the operation the patient was in considerable shock, she was cyanotic and the pulse was hardly perceptible. Under appropriate treatment she improved somewhat, but died about ten hours after operation, giving the clinical picture of a severe case of pancreatitis which has not been operated on. The impression we had was that operation had been carried out too late.

Etiology of Acute Pancreatitis.—About 300 years ago the pancreas was still considered to be simply a pad for the support of the stomach and the protection of the large vessels. In 1642, Wirsung succeeded in finding the excretory duct and since then this organ has been known to be a gland, but it was not until the middle of the last century that Claude Bernard emphasized the importance of the pancreatic juice in the digestive process. The first reliable reports concerning diseases of the gland date back to this period, but only during the last twenty or thirty years has the field of pancreatic diseases been opened to the surgeon.

Acute pancreatitis is one of the diseases that has become generally known more recently. For our first knowledge concerning fat necrosis we are indebted to Balser, who in 1879 wrote his famous article on "Fat

Necrosis, a disease of man often ending fatally". However, he did not recognize the intimate relation between this condition and pancreatic disturbance, but considered it an idiopathic disease. He performed many autopsies to study the condition and frequently found these little opaque yellowish-white spots in the region of the pancreas in people who had died of a variety of diseases. He called attention to the fact that nearly all patients in whom fat necrosis was found were over forty years of age and were usually well developed, rather adipose subjects. He also looked for this condition in animals, especially those rich in fat, and frequently found fat necrosis in the pancreas and its vicinity in Hungarian pigs without any disturbance of their general well being. In animals poor in fat and in German pigs he rarely found it. Williams made similar observations in two hundred American pigs examined. After Balser's reports considerable attention was paid to this subject, but whether fat necrosis or pancreatic disease was the primary affection remained in dispute for a long time. Fitz deserves the credit for first emphasizing the primary affection of the pancreas. As a result of his studies and a review of the literature he expressed the conviction that fat necrosis is always secondary to disease of the pancreas. In the course of time various theories concerning the nature of the disease were advanced. The infectious theory was the most popular and still has many adherents. Heava believed the disease depended on the entrance of hydrochloric acid into the excretory duct. Rolleston on the other hand believed it to be a tropho-neurosis. As a result of extensive animal experiments the theory that the disease is due to the activity of ferments probably has the largest number of adherents to-day.

Several of the symptoms, and also the course of the disease, seems to point to infection as the cause. Microörganisms have been found by several authors in the pancreatic tissue itself, as well as in venous thrombi in the gland and in its surroundings. They have also been found in the peritoneal exudate and in the vicinity of the fat necroses. But as bacteria are often found in dead bodies, several authors have tried to isolate them during life, and they have tried to induce fat necrosis by means of the injection of bacteria. In several instances they succeeded in producing typical fat necrosis by the injection of bacteria into the pancreas directly, or into its excretory duct. The most prominent present advocates of the infectious theory, are Deaver and Pfeiffer who have contributed extensively to this subject. They have found clinically that infection may spread to the pancreas by way of the lymphatics from an inflamed gall-bladder, a gastric or duodenal ulcer, or chronic appendicitis, and they have shown experimentally the possibility of such extension. Barber has also in a recent article expressed the belief that infection is carried from a cholecystitis through the portal vein to the liver and that it then descends through the lymphatics to the lymph-nodes and the pancreas, producing milder forms of pancreatic lymphangitis. But in spite of these findings the infectious theory does not seem to explain the condition. No acute inflammation has been demon-

strated in the fat necroses, and bacteria have been but rarely found in the necrotic foci or their vicinity. Truhart does not believe in this theory and states in his monograph that the pancreas of men is not susceptible to bacterial invasion, and that the excretory duct but rarely allows bacteria to enter. Archibald and Gibbon also do not believe in the lymphatic origin, and in commenting on Deaver's work dismiss it by saying that it is still under discussion, and in any event is hardly susceptible of absolute demonstration, it lacks as yet experimental proof, depends upon purely clinical inferences, and goes on the whole contrary to the rules of pathology.

Our principal knowledge concerning this acute affection has been gained by animal experiments, because in man it often runs such a short and stormy course that long observation is impossible, and in addition to that the situation of the gland precludes easy access. We have tried in a few cases to learn something by means of the duodenal tube, but the incessant vomiting made it impossible to keep the tube down. In order to carry out successful animal experiments it is necessary to produce conditions which may appear spontaneously in the human body. Many authors have taken this into consideration and they have proved in this way that fat necrosis may be produced by local disease of the pancreas such as artificial inflammation, stagnation of its secretion, flow of pancreatic juice into the retroperitonel fat, etc.

Langerhans made a suspension of fresh pancreas in distilled water, injected it into fatty tissue and produced fat necrosis. He first called attention to the fact that their development depended on the splitting of the fat into its component parts, glycerine and fatty acids. Hildebrandt produced fat necrosis in animals by means of steapsin, and hemorrhages by means of trypsin. They were brought about by simple stasis of the secretion or of the return flow of blood, or by laceration of the pancreas. These experiments were later repeated by Williams, who tied off a part of the pancreas and lacerated it and produced fat necrosis in cats. We also have had occasion to repeatedly note extensive fat necrosis in dogs which died suddenly after operations on the duodenum. They were probably the result of lacerations of the pancreas with entrance of the juice into the retro-peritoneal tissue.

Of greatest importance for the theory of ferment action was finally the demonstration of fat splitting ferments in the necrotic foci, by Flexner. He proved that pancreatic juice may flow into the peritoneal cavity without producing inflammation, and further that sterile pancreas or pancreatic juice may produce fat necroses. As the result of his experiments he came to the conclusion that infection as such played no rôle at all, or only a minor one. The final result of his work led him to state that fat splitting ferments can be demonstrated in fat necrosis at certain times, especially in the early stages, and that later they may disappear entirely. Though it is not possible to state positively that steapsin is the direct cause of fat necroses, it most probably is, because it can always be found in them and it is not present

in normal fat. The permeation of pancreatic juice into the peri- and para-pancreatic tissue is the cause of fat necrosis, and this permeation is made possible by laceration of the pancreas or circulatory disturbances.

Hess showed that the injection of a few c.c. of sterile olive oil into the duct of Wirsung would produce necrosis of the pancreas and surrounding fat. The animals died under similar circumstances as human beings, and the autopsy findings were identical. Injection of glycerine did not produce these results. From these findings the following conclusion was drawn : Contact of fat and pancreatic juice in the duct splits up the fat and produces soap. This soluble soap passes from the duct into the pancreatic tissue, destroys it and opens the way for pancreatic juice into the surrounding fat. The latter is split and necrosis develops and forms the nucleus for a spreading necrotic process. These experiments are more successful at the height of digestion because at that time the gland is filled with juice.

Similar conditions may play a rôle in the human body, because at the height of digestion the duodenum contains much fatty material, and the pancreas much juice. Compression of the duodenum may be brought about by a full stomach or by muscular action, and some of its contents may enter the common bile duct and the duct of Wirsung. Such an occurrence is more easily conceivable in the presence of a dilated papilla, which may have been injured by the passage of a gall-stone. In this connection it is interesting to note that the onset of the symptoms of acute pancreatitis in many patients is definitely given at the height of digestion, a few hours after a heavy meal, But even without injury to the papilla one must admit that relaxation of the sphincter may at times allow the entrance of duodenal contents. We do know that sphincters guarding other viscera under certain circumstances allow regurgitation, as for instance the pylorus, the ureters, the ileocæcal valve, etc. It is also commonly assumed that inflammation may spread from the duodenum to the bile ducts and produce catarrhal jaundice.

Other experiments consisted in ligating the excretory duct of the pancreas in dogs at the height of digestion. As a result of the tension produced in the ducts they ruptured and allowed extravasation of pancreatic juice into the surrounding tissue, producing typical necrosis. The same technic employed in starving animals did not produce necrosis, the secretion of an active juice was inhibited, and fibrosis of the gland initiated. In a human being such stagnation of juice may develop as the result of spasm of the papilla or by the incarceration of a gall-stone in that part of the common duct where it passes through the pancreas and where it is able to compress the duct of Wirsung. Several such cases have been reported.

The injection of bile into the excretory duct likewise produces necrosis of the pancreas and fat, probably the result of the activation of steapsin by the bile, and owing to the combination of pancreatic juice with the fatty substances contained in bile. That this mode of development is possible in man due to the incarceration of a gall-stone at the papilla has been proven chiefly by Opie and Korte.

As the result of experiments and clinical observation Archibald and Gibbon have come to the conclusion that in cases of acute pancreatitis bile most likely flows into the pancreas, and they believe that three conditions must be present to make this possible. (1) There most likely is a change in the composition of the bile, increasing the proportion of bile salts. (2) There must be undue resistance, perhaps amounting to spasm of the common duct sphincter. (3) There must be an abnormal rise of pressure in the biliary system behind, either in the gall-bladder or in the common duct.

Mann and Giordano have undertaken the most recent and very extensive research into the problem of the causes of pancreatitis. Unfortunately only a part of their results are so far available. Their first publication deals with the bile factor in the production of the disease. They have studied this subject experimentally, and on the basis of anatomical findings in man, especially the relation of the bile duct to the pancreatic duct. After the most painstaking observations they have come to the conclusion that although reflux of bile into the pancreatic duct may occasionally take place, this cannot be considered the cause of acute pancreatitis.

Bearing in mind all the experimental data submitted so far, as well as the reports of careful clinicians, and in that light review our own six cases regarding their etiology, it would seem.

1. That infection by means of lymphatic extension played no rôle in any of the cases. Had there been an infection in any other organ leading to such an acute condition in the pancreas there should have been symptoms of such an infection. But that was not the case. All patients were stricken suddenly, while apparently in excellent health, and the symptoms were at once referred to the epigastrium or at least to the upper abdomen. In none of the patients was there early temperature or rise in pulse. The blood-count was high, but that may be so in conditions not strictly inflammatory in character. In addition to this no acute inflammatory signs were observed in any organ at the time of operation, and the cultures taken from the peritoneum and the retroperitonal tissue as well as from the gall-bladder, were all negative. The tissues from Case I, which came to autopsy, showed no acute inflammation except in the fatty tissue surrounding necrotic areas, where it formed a protective barrier.

2. That regurgitation of duodenal contents may have played a rôle. Such regurgitation is theoretically possible, unfortunately it can neither be proved nor disproved. In three patients the onset of acute symptoms occurred at the height of digestion, a few hours after a heavy meal, when conditions for regurgitation and for the development of pancreatitis were favorable.

3. That the gall-bladder and bile probably in some way were connected with the development of acute pancreatitis. In five patients gall-stones were found and in the sixth a diseased thickened gall-bladder. Just what the relationship was, whether bile actually entered the pancreatic duct, or whether a gall-stone or spasm of the sphincter temporarily obstructed it and caused

increased pressure, with subsequent rupture of the pancreatic duct, is impossible to state positively. It does appear though that something took place to affect the entire gland at once, for the onset in all cases was acute and very severe. The fact that in two patients jaundice was noted, also speaks for the close association of the two conditions. In Case III, the diagnosis of cholecystitis rested partly on the fact that she was jaundiced, and in Case IV, in addition to the icteric tinge of the skin, bile was found in the urine, and the bile color index of the blood was considerably increased. Granted that bile may enter the pancreatic duct under certain conditions, and this is admitted even by Mann and Giordano, the next question is whether normal bile is able to produce acute pancreatitis. The two last named authors practically deny this while Archibald and others have experimental proof that it is possible. This question however, is of not so much importance clinically, because in the patients in whom gall-bladder disease and acute pancreatitis have been found associated the bile has not been normal. It may have been sterile, but its chemistry had been altered. Archibald states that in infected bile it is pretty certainly not the bacteria acting in their infecting capacity that cause the pancreatic lession, but rather the chemical change in the bile produced by the action of bacterial growth. This is the conclusion I have also reached as the result of observations in my patients.

That in Case I, the destructive process started in the pancreatic duct is evident from the nature of the lesion found at autopsy. In the other cases no doubt a similar progression took place, leading to complete recovery of the tissue in two patients and necrosis of the tail portion in two others. Only a process acting through the duct or the circulation can affect an entire gland so suddenly and so severely. None of the patients had any inflammatory symptoms preceding the acute onset, and even after the onset some time elapsed before temperature and increased pulse developed. This increase is probably the reaction to a chemical irritant, and not the result of a primary inflammation. Cultures taken during the acute stage from the peritoneal fluid, the retroperitoneal exudate and the gall-bladder showed no organisms. In each case the gall-bladder showed chronic inflammation, but no acute signs.

Any other factors sometimes named in the etiology, such as pregnancy, alcoholism, obesity, etc., are probably simply contributing factors, in that they bring about conditions favorable for the development of chemically altered bile or for spasm of the sphincter and congestion of the liver. It is well known for instance that during pregnancy, women have a high cholesterinæmia and that gall-stones are apt to form. It is then the presence of the gall-stones and the induced chronic cholecystitis, which is directly responsible for producing pancreatitis, and not the pregnancy. In Cases III and V, symptoms dated back to their last pregnancy and in Case IV, to an abortion. In alcoholism it is probably the hyperacidity and congestion of organs which bring about conditions favorable for spasm of the

sphincter, and at the same time congestion of the liver, which may raise the pressure in the biliary system to such a degree that bile passes over into the pancreatic duct. Mann and Giordano state that in only a small percentage of human beings is it anatomically possible for bile to pass over into the pancreatic duct. Granted this is so, one may answer that by saying acute pancreatitis is a rare disease as compared with gall-bladder disease, and that it is only in that small percentage of patients in whom the necessary anatomic, physiologic and pathologic factors are present, that acute pancreatitis develops.

Symptoms and Physical Signs—Pain is by far the most important and persistent symptom. It is at once severe and overwhelms the patient with the suddenness of its onset. It is colicky in character, is situated in the upper abdomen, in most patients in the epigastrium, in others more to the right or left. It frequently radiates to the back and shoulders. One patient described a feeling as of a girdle pain, constricting her entire upper abdomen. The pain is of such severity that patients are unable to rest, they moan and toss about and beg for relief. Sometimes pressure eases the acuteness, at other times morphin, but as soon as that wears off, the pain returns. It may come somewhat intermittently (Case IV), as it does in intestinal obstruction, but usually it is steady and never lets up.

Vomiting is the next most important symptom, and in the more severe cases recurs every few minutes. It is distressing to see such a patient in pain retch and strain trying to bring up a small amount of fluid. Vomiting may continue very frequently for a day or more and then stop, or it may continue until operation or death. In one of the patients it lasted for two weeks. The vomitus is usually yellow in color, has no abnormal odor and no other unusual characteristics. After vomiting ceases, hiccough and retching may continue as evidence of irritation in the upper abdomen.

Collapse symptoms are frequently present, they are more or less severe depending on the intensity of the attack. They were observed in three of my patients, and indicate severe disturbance as evidenced by the fact that two of them died. In the very severe cases with hemorrhage or extensive necrosis cyanosis may be present and death may occur within a few hours or days.

The physical signs depend on the stage during which a patient is seen and on the severity of the attack. Most patients now come under observation in the first few days and during this period the signs point to some acute lesion in the upper abdomen. What has struck me as most interesting is the lack of physical signs as compared with the severity of the symptoms. This has been true in two of my patients to such a degree that the house staff ventured to suggest that they were simply neurotic. At the time they came under observation four of my patients had no rise in temperature and the pulse rate was not increased. The abdomen was only slightly distended and there was slight rigidity or none at all. The most marked sign was exquisite tenderness over the entire upper abdomen, perhaps

most marked just above the umbilicus and to the left of the median line. Such exquisite tenderness with only slight rigidity or none at all, and the lack of other signs has seemed to me to be additional evidence that we were not dealing with an inflammation due to infection, but rather with a chemical irritation of the peritoneum. Other aseptic peritoneal exudates, blood from an ectopic for instance, will give similar findings.

The blood-count in the patients in whom it was done during the early stage, showed leucocytosis with an increase in the polymorphonuclear count. Icterus was present in two patients early in the disease, and was therefore probably not due to pressure of the enlarged pancreas on the common bile duct, but rather to the same underlying condition which was responsible for the pancreatitis, namely temporary obstruction at the papilla, either in the form of a stone or spasm.

The diagnosis, though often doubtful, is made with increasing frequency, as judged by the more recent literature. In my series we were fortunate enough to make it with reasonable certainty in Cases I, IV, V, and VI, while in Case II, it rested between perforated gastric ulcer and pancreatitis and in Case III, we did not consider it at all, but made a diagnosis of cholecystitis. In this last case I believe the diagnosis could have been made, had we paid more attention to the left-sided pain and tenderness. The history is important, because most patients have had previous attacks of upper abdominal pain, which may have been mild attacks of pancreatitis or were caused by the existing cholecystitis or cholelithiasis. All my patients gave a history of previous attacks, and while some stated they had been different than the one for which they were admitted, others were under the impression that they were the same type but milder in character. We do know that the cases operated upon vary a great deal in regard to severity of symptoms and extent of lesion and it is therefore not difficult to believe that some attacks may be so mild that they subside without coming to operation. I have been interested in this question for some time and have made it a point to look for fat necrosis when operating upon the gall-bladder or stomach, but so far I have encountered only one patient, a man with a penetrating duodenal ulcer, who showed fat necroses without having symptoms referable to them or to pancreatic disease. Several years have elapsed since that operation and the patient has remained well. In one of my patients, Case IV, after making a positive diagnosis of acute pancreatitis, I was willing to test the question whether spontaneous cure was possible, but the symptoms steadily grew worse and operation became imperative. Acute pancreatitis is most often mistaken for cholecystitis, for peritonitis from a perforated viscus, or for acute intestinal obstruction, and it will not always be possible to differentiate between them. Bearing in mind however, particularly the difference between the intensity of the symptoms, and the slight distention of the abdomen, slight or no rigidity and the absence of obstipation, should at least make one think of acute pancreatitis, especially when the patient has been apparently healthy, is obese, and the attack has come on soon after

a heavy meal. Laboratory examinations are of little value. The vomitus and the fæces show nothing characteristic, and the blood examination will at most indicate that an acute condition exists. The urine may show a small amount of sugar, the presence of which may make us think in the right direction.

Treatment.—The general opinion among surgeons today is that the disease is a surgical condition, no matter during which stage it comes under observation. A study of the literature, and especially an excellent article by Körte show conclusively that operation during the early stage offers the best chance. In order to determine what we want to accomplish by an operation we must know what are the most likely causes leading to a fatal outcome. There are various opinions on this subject and it is possible that several causes work together. Some authors attribute death to shock or hemorrhage, others believe it is due to the sudden throwing out of function of an important gland. The most popular theory is that it is due to poisoning of the body by decomposition products of the pancreas or a toxæmia produced by fat necrosis. That the exudate in the peritoneum and retroperitoneal tissue is toxic, and is chiefly responsible for the general symptoms is demonstrated by many cases in which nothing was done but to sponge out this exudate, and recovery followed. To accomplish this is therefore one of the objects of the operation. It is also believed that the tension in the pancreas and peri-pancreatic tissue is partly responsible for necrosis of the gland. Relief of this tension during the early stage of the disease may therefore inhibit or limit the amount of necrosis. Such relief of tension can be brought about by splitting the fat overlying the pancreas or even entering the pancreatic tissue if areas of softening are present. Hemorrhage may be controlled or at least the hemorrhagic exudate may be drained instead of allowing it to spread into the retroperitoneal tissue, for it is probably the pressure on, or the penetration of the sympathetic ganglia by the exudate, which produces shock. It is seen therefore that an early operation is able to counteract almost any of the possible causes of death.

The best approach is through the median or right rectus incision. Large sponges are at once inserted to absorb exudate and prevent spreading it into the lower abdomen which is usually not involved. The exudate is then sucked or sponged out and the upper abdomen explored. I have approached the pancreas in my cases through the gastro-colic omentum, it gives good access and allows of good subsequent drainage. If the pancreas does not look or feel badly diseased, one may be content to insert gauze tampons into the retroperitoneal space for drainage. Usually however it is well to split the fat overlying it, bluntly and then insert the gauze tampons directly to the pancreas. The steps so far carried out have removed the free exudate, have relieved the tension in the gland and by draining the retroperitoneal tissue have produced favorable condition for carrying the toxic exudate outward, instead of allowing it to spread further into the tissue. One may

end the operation here, or devote some attention to the associated conditions. All of my cases had gall-bladder involvement. The question arises what to do with these associated lesions. My belief is, that if access is good and the patient's condition warrants it, one should remove the gall-bladder at once. The reverse being the case, it is better to postpone the gall-bladder surgery to another date. What I actually did do in the five patients on whom I operated was to leave the gall-bladder alone in two cases, both recovered, but one subsequently had to be operated on. In one patient I removed the stones and drained the gall-bladder, she also recovered. In the other two patients I removed the gall-bladder, one recovered and one died. However, death was not due to this, but to the fact that the patient had a most extensive hemorrhagic type of pancreatitis of which she died a few hours after operation.

Prognosis.—The prognosis depends on the amount of damage done, the intensity of the toxæmia, and the time at which operation is performed.

There is ample evidence that patients may recover from the acute attack without operation and develop one of the late sequelæ, such as pseudocyst, abscess, or necrosis. The treatment of all these conditions is surgical. I believe that if the entire pancreas is at once extensively involved as my Case I was, there is very little hope for the patient, unless drainage and relief of tension are instituted very early. If only a portion of the gland is involved, the prognosis is better, especially if the tail instead of the head is involved. In my two patients who discharged a necrotic slough about three weeks after the onset of the acute disease, it was the tail-end of the gland, which was discharged.

Once operated on for pancreatitis, are the patients likely to develop another attack? Theoretically one would say that if the predisposing factors continue to operate, there is reason to believe that subsequent attacks may follow. It will therefore be well to pay attention to the associated gall-bladder disease, either at the time of operation, or subsequently. Several recurrences have been reported in the literature. My own patients have remained well, seven, two, two, and one and a half years after operation.

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POST-OPERATIVE STRICTURE OF THE COMMON BILE DUCT

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The bile ducts are lined by a single layer of tall columnar epithelium which is supported by a rather thick layer of elastic tissue. Then comes a layer of muscular fibres, and finally fibrous tunic. Scattered through the wall of the duct and communicating with its lumen are numerous simple tubular glands. The lining epithelium is possessed of extraordinary powers of regeneration and quickly covers any break in its continuity; in fact it grows into and lines a new duct constructed from other tissue, as was shown experimentally by Horsley. Injury to this layer of epithelium, whether by trauma or infection, probably is not of much consequence. The layer of elastic tissue which might be called the submucosa contains the real strength of the duct wall and permits of wide dilation. Injury to the submucosa by trauma or infection, and its replacement by fibrous tissue, represent the basic process of the pathology of stricture formations. A stricture can be lined by normal epithelium.

Instances of stricture of the common duct, independent of disease of the gall-bladder or stone in the duct, have been reported, but are exceedingly rare. Many cases spoken of as post-operative stricture are improperly designated because in the majority either the stricture itself, or the lesions which later developed into stricture, was present at the time of operation. True post-operative stricture is a sequel of operation in the same sense as post-operative hernia. According to this conception of the condition, the greater percentage of strictures of the common duct following operation on the gall-bladder or ducts, is caused by operative trauma.

We have collected from the records of the Mayo Clinic forty-eight cases of stricture of the bile ducts, and from this group we have selected for the present study ten cases in which the symptoms appeared in not less than six months after operation on the biliary tract. The interval of six months was arbitrarily chosen, because a lesion existing at or before operation and likely to develop into stricture would probably become manifest as a stricture within six months. It is possible that some of our cases are not cases of true post-operative stricture, as we have previously defined the term.

Injury to the ducts during cholecystectomy is an accident dreaded by every surgeon. Immediate recognition of the mistake is mortifying, but not necessarily alarming because repair usually can be easily and satisfactorily made. When the injury escapes the attention of the surgeon, nature proceeds with repair, but the handicap may be too great and stricture is then prone to follow; a persistent biliary fistula or intense jaundice signal the onset of serious trouble.

Undoubtedly many instances of injury to the ducts occur which go on to spontaneous repair, and never give rise to symptoms.

It is difficult to determine what degree of injury is required to produce a stricture with symptoms. In many strictures which we have seen there was a lumen that permitted the passage of bile, but periods of recurring inflammation in the stricture gave rise to attacks of biliary obstruction, as evidenced by colic and jaundice. Occasionally there is a stone above the stricture which may be the cause of intermittent obstruction.

Many factors which predispose to injury of the ducts are well known, and it will suffice here merely to mention a few of the more important, such as anomalies of the ducts and vessels; traction on the pedicle of the gall-bladder, especially when removal is begun at the fundus, thus producing angulation of the common duct; and blind grasping with forceps for a bleeding cystic artery. The crux of the situation is failure accurately to identify the cystic duct. If the presence of inflammatory exudate around the pedicle of the gall-bladder or other conditions make definition of structures doubtful, it is certainly an act of better judgment merely to drain the gall-bladder rather than to jeopardize the welfare of the patient by persisting in efforts to carry out cholecystectomy. When the gall-bladder is distended by fluid or stones, or both, the region of the cystic duct is often inaccessible. A complete operation, that is, cholecystectomy, can be safely carried out in many such cases by first emptying the gall-bladder with the aid of a trocar or scoop.

Oddi was the first to show that the stump of the cystic duct dilated after cholecystectomy. His observation was confirmed by Haberer and Clairmont. Eisendrath and Dunlavy conducted similar experiments with identical results, and they also report two clinical cases in which small calculi were found in the stump of the cystic duct, and presumably were responsible for the patient's symptoms. Hartman, Smyth, and Wood have recently reported, as the result of their experimental work, that the stump of the cystic duct hypertrophies, not merely dilates, and takes on the appearance of a new gall-bladder: nature's compensation for the one removed by the surgeon. In the foregoing experiments particular efforts were made to leave as much of the cystic duct as possible, and the results have been used to influence the surgeon to remove the entire cystic duct flush with the common duct when performing cholecystectomy. We believe that this is misguided enthusiasm and will result in more harm than good. It is unwise, for obvious reasons, to leave the entire cystic duct, perhaps with a portion of the neck of the gall-bladder, after cholecystectomy, but, on the other hand, efforts to remove the entire cystic duct often result in excision of an oval piece from the wall of the common duct or inclusion of a portion of the wall of the duct in the ligature; a stricture usually follows. To leave a small stump of cystic duct sufficient to insure the integrity of the common duct is, we believe, the safest procedure, and one which will rarely give rise to future trouble. The cystic duct usually is involved in disease of the gall-bladder, and the stump would, therefore, be unlikely to undergo hypertrophy.

Incision of the common duct for removal of stones, for drainage, or for exploration, is rarely a cause of stricture. Cases in which cicatricial stenosis followed operations on the duct are usually ones in which calculi were removed, and in all probability the damage to the duct wall incident to the presence of calculi is the cause of the stricture. In four of our cases in which stricture developed, stones were removed from the common duct, and in three of these a biliary fistula persisted for several months; this was probably an indication of beginning stenosis of the duct. Incision of the duct is usually made in its long axis because it affords the best exposure. Transverse incision is probably equally safe, so far as stricture formation is concerned, but it is not so useful. Drainage of the duct by the T-tube, introduced by Kehr, is still used by Deaver and others with satisfactory results. Catheter drainage by the Robson method is also satisfactory.

It is difficult to explain the occurrence of post-operative stricture when cholecystectomy has been performed for simple chronic cholecystitis without stones and no apparent disease of the ducts, and when subsequent operation shows a stump of cystic duct with no evidence of injury to the major duct. A very probable explanation, we believe, is a localized infection of the glands of the wall of the duct which has resolved with the formation of fibrous tissue.

Symptoms of Stricture.—The time of onset of symptoms is variable. One patient was well for three and one-half years following cholecystectomy, then began to have colics with jaundice. It is possible that this lesion had no connection with the former operation. It is a significant fact that four patients stated that, following removal of the gall-bladder, a biliary fistula persisted for several months. There was probably partial obstruction of the duct at that time. When the fistula closed the patients remained well for several months, and then developed symptoms of obstruction. One might suppose that a stricture, when it begins to cause trouble, would do so by producing more or less complete biliary obstruction. This was true in only four of our patients. One had constant jaundice and attacks of colic; one had a total biliary fistula; one had intermittent jaundice with opening and closing of a biliary fistula; and one had permanent jaundice.

The usual symptoms are those of intermittent biliary obstruction, that is, attacks of colic with jaundice such as occur with common duct stone. Four patients had attacks of severe colic and jaundice; and one had intermittent jaundice without pain. There is an apparent analogy with the symptoms of urethral stricture. Probably a low-grade chronic infection is associated, which causes temporary œdema sufficient to close the lumen of the stricture. The harmful effect of stricture is, of course, interference with free drainage of bile and consequent impairment of hepatic function, the result of back pressure. It is also probable that the liver may be considerably damaged before obstruction is manifested by clinical signs. An important factor in the surgical risk of these patients is the long duration of symptoms of biliary obstruction before they seek operative relief. One patient was deeply jaundiced for three months; one had had a biliary fistula and attacks of cholangitis with jaundice for nine

months, and one had had attacks of cholangitis with jaundice for eight months. The remaining seven patients had had symptoms of intermittent biliary obstruction for from two to forty-two months.

The usual location of the stricture is at the juncture of the cystic with the common duct. The involvement of this area might be due to the fact that it is the most common site of injury; it is the least dilatable portion of the duct; and it is very often involved by extension of inflammation from the gall-bladder and cystic duct. The stricture itself when encountered at operation appears as a fairly definite area of constriction from 0.5 to 1.5 cm. in length. It may be tubular and annular, or it may affect only a portion of the circumference of the duct. The lumen is definite, but very small. The duct above the stricture is markedly dilated, whereas the portion below often maintains its normal appearance.

Operation.—The most difficult part of the operation is to locate and identify the duct. The adhesions are usually very dense, but this may be favorable, since the operation can often be performed entirely extraperitoneally. The operation is most tedious, requiring time and patience. When a fistula exists, it leads directly to the stricture. Otherwise it is advisable to work through an incision close to the median line and to follow the gall-bladder fossa from its notch in the edge of the liver. As the site of the duct is approached, the use of a syringe and needle is a great help in the identification of the structure. It may be difficult to distinguish between the dilated duct and the portal vein. If patients are jaundiced or very sick, the best plan is to secure drainage of bile to the outside, thereby favoring resumption of hepatic function and subsidence of cholangitis. The stricture is divided in the direction of the duct, and a T-tube inserted, which should be kept in for several months. Permanent relief will be obtained in many cases. Sometimes, stenosis recurs when the T-tube is removed. The methods of operative procedure employed in this series will be found in the reports of the cases.

Results of Operation.—The condition, owing to its nature, entails grave surgical risk, yet without operative measures the outlook is absolutely hopeless. Patients who recover from operation usually have mild attacks of colic, with or without jaundice, for several months, or sometimes several years, and then completely recover. The long duration of biliary obstruction or infection before operation often results in biliary cirrhosis which is sometimes the basis for post-operative symptoms. At best, these are very difficult and tedious cases, and present what is probably the most formidable lesion of the upper abdomen.

SUMMARY

Post-operative stricture of the common duct is usually the result of operative trauma. It may also follow localized infection or necrosis of the wall of the duct. The symptoms may be those of permanent or intermittent biliary obstruction, and often suggest stone in the common duct.

The patients are very ill and are grave surgical risks because of jaundice,

cholangitis, and impaired liver function. The site of the stricture is usually at the juncture of the cystic and common ducts.

Operation should provide biliary drainage and restoration of the normal course of the bile.

Results of operation are fairly satisfactory, considering the hazard and technical difficulty, and the otherwise almost hopeless nature of the condition.

ABSTRACTS OF CASES

CASE I.—(A51191). A woman, aged fifty years, had had hepatic colics, sometimes associated with jaundice, for fifteen years. Cholecystectomy and removal of stone from common duct had been performed elsewhere seven years before. She remained apparently well for three or four years, and then had had a return of the attacks of colic and jaundice almost daily for the last month. Four months before, she had had several gastric hemorrhages and passed blood in the stool.

Examination revealed that the liver was markedly enlarged. At operation, April 3, 1911, the common duct was found to be strictured at a point where the gall-bladder had been removed. The duct was incised for 1.9 cm. and scoops passed into the duodenum. A McGuire drain was inserted. The patient died seven days later.

CASE II.—(A39253). A woman, aged thirty-one years, had had attacks of hepatic colic for two years. Cholecystectomy for chronic cholecystitis without stones had been performed at the Mayo Clinic, September 10, 1910. The patient was well for two years, then had recurrent attacks of colic and much soreness over the right upper quadrant of the abdomen. Jaundice followed one attack.

A second operation was performed June 13, 1913, for stricture at the juncture of the common and cystic ducts. The stump of the cystic duct was split down into the common duct, and a Robson catheter drain inserted. The symptoms were relieved for about one and one-half years when there was a return of pain and soreness in the right upper quadrant, gastric distress, chills and fever, and occasional jaundice. A diagnosis was made of cholangitis with partial biliary obstruction.

A third operation was performed April 3, 1915, for recurrent stricture of the common duct. The duct was split longitudinally from the dilated portion down through the area of constriction. Forceps and scoops were passed into the duodenum. A T-tube was inserted, one end going down into the duodenum, the other extending 2.5 cm. up into the hepatic duct. The patient was examined in the Clinic seven and one-half years later and was perfectly well.

CASE III.—(A39393). A man, aged fifty-six years, had had a cholecystostomy for stones elsewhere seven years before. A short time after the operation he had had attacks of colic; he was then in good health until two months before examination at the Clinic, when, following a severe colic, a fistula opened in the old scar and drained mucus. He had not noticed that the stools were white.

Operation was performed at the Clinic August 26, 1915, for stricture of the common duct, at the juncture of the cystic ducts. The gall-bladder contained stones, and there was one stone in a diverticulum of the gall-bladder which, by pressure on the common duct, had produced the stricture. The common duct below the stricture was normal. Cholecystectomy was performed. The stricture was incised and a T-tube inserted. The patient died two days after the operation, from renal insufficiency.

CASE IV.—(A125271). A woman, aged forty-six years, had begun to have attacks of sick stomach, with chills and fever, jaundice, clay-colored stools, and a feeling of pressure in the epigastrium in November, 1913. Operation had been performed elsewhere May 9, 1914, and an abscess near the common duct drained. A second operation was performed elsewhere June 17, 1914, and a stone removed from the ampulla of Vater. This was followed by a biliary fistula which was constant, and present at the time of her admission to the Clinic. She also had frequent attacks of chills, fever, and jaundice.

At operation at the Clinic March 1, 1915, the patient was deeply jaundiced; the gall-bladder and duodenum were adherent, and a cholecysto-duodenostomy was performed. There were no stones. The biliary fistula was not disturbed. The patient had fair health for three months after operation; then had attacks of chills, fever, and jaundice with clay-colored stools. At operation at the Clinic September 30, a stricture of the common duct and a few small stones in the hepatic duct were found. The stricture was excised and the hepatic duct united to the duodenum, the anastomosis being made over a small rubber tube.

The patient was examined in the Clinic, April 18, 1917. She had been apparently well for ten months after the last operation, and then began to have slight attacks of chills and fever. Jaundice was present. A diagnosis was made of biliary cirrhosis. A röntgenogram showed that the tube used for the anastomosis was still in place. The patient died July 21, 1921.

Case V.—(A142876). A woman, aged fifty-six years, had had gall-stone colics for fifteen years, and had been jaundiced in several of the attacks. Operation was performed elsewhere in June, 1914, and the gall-bladder drained for empyema. Jaundice, which was present at the time of operation, did not clear, and at a second operation six weeks later a stone was removed from the common duct. The jaundice cleared, but a biliary fistula persisted for five months. About two or three months after closure of the fistula, attacks of colic with jaundice and clay-colored stools recurred.

At operation at the Clinic, October 12, 1915, a very tight stricture of the common duct was found. The hepatic duct was united to the duodenum over a T-tube. The patient was not relieved, and died four months later.

Case VI.—(A42032). A woman, aged forty years, had had a cholecystectomy performed elsewhere two years before. She was well for six or eight months, and then had a return of colics similar to those before operation. There was no history of jaundice.

At operation at the Clinic, September 29, 1915, the pelvis of the gall-bladder and the cystic duct, which had not been removed, were found to contain stones. There was a stricture at the juncture of the hepatic and cystic ducts, and an area of about 1.25 cm. had been removed from the anterior wall of the duct. The stump of the gall-bladder and the cystic duct were dissected out. A piece of the cystic duct with its mucosa was saved and sutured over the defect in the wall of the duct, thus uniting the hepatic and common ducts. Scoops had been passed into the duodenum. Seven years later, the patient reported that she was perfectly well.

Case VII.—(A75971). A woman, aged forty-one years, had had attacks of severe epigastric pain for twelve years. She had never been jaundiced.

At operation at the Clinic in November, 1912, a thick, white gall-bladder was removed. There were no stones. The patient remained well for three and one-half years, and then began to have attacks of severe epigastric pain radiating to the back. Jaundice was present on several occasions.

A second operation was performed April 15, 1917, which revealed stricture of the common duct at the juncture of the cystic duct. The stricture was incised longitudinally, and then sewed transversely over a T-tube. The patient continued to have attacks of colic for two years after removal of the tube, but her last report five and one-half years after operation stated that she was in good health.

Case VIII.—(A150805). A woman, aged forty-two years, had had attacks of gall-stone colic for three years; there was no history of jaundice. The gall-bladder containing stones had been removed elsewhere one year before. The patient remained well for about seven months, and then had a severe attack of colic followed by jaundice which persisted.

Operation at the Clinic, February 12, 1918, disclosed a stricture of the common duct at the juncture of the cystic duct. The duct below the stricture was apparently normal. There was no bile in the ducts. The stricture was incised and a T-tube inserted. The patient returned six months later and, while undergoing examination in the Clinic, developed an acute abdominal condition which at operation proved to be abscess of the liver. She was well for four months after this operation, and then began to have

recurring attacks of colic with jaundice and occasionally with fever. When she presented herself for examination there was definite jaundice.

At operation September 23, 1919, a stricture of the common duct was found. The side of the dilated duct above the stricture was united to the duodenum, the anastomosis being made over a short rubber tube. A report three years later contained the information that the patient vomited the tube one year after the operation. She is in good health, but occasionally is slightly jaundiced.

CASE IX.—(A39301). A woman, aged forty years, had had a cholecystectomy for cholelithiasis and appendectomy elsewhere in 1914. A stone was removed from the common duct elsewhere in 1917. This was followed by a biliary fistula for two months. Seven months after the fistula closed, the patient began to have attacks of hepatic colic with jaundice, sometimes with chills and fever.

Operation was performed at the Clinic, May 29, 1918, for stricture of the common duct at the juncture with the cystic duct. The stricture was dilated by opening the duct below it, and a Robson drain passed up through the stricture. Two months later the patient again began to have attacks of colic, occasionally with jaundice.

At operation performed June 19, 1919, it was found that the stricture had recurred at the former site. It was incised and the duct reconstructed over a T-tube. The tube was kept in place for three months, and following its removal, constant pain developed over the area of the liver, but no jaundice.

A third operation was performed July 14, 1920. The stricture had again recurred. It was incised and the duct reconstructed over a small rubber tube. The patient remained well for two years, but now reports a constant dull pain over the area of the liver.

CASE X.—(A315910). A woman, aged twenty-six years, had had a cholecystectomy for stones elsewhere three years before. This was followed by a biliary fistula which drained intermittently for several months and then remained closed. The patient had remained well until about eight months before, that is, about two years following the cholecystectomy. She then began to have attacks of chills and fever, vomiting and slight jaundice. There were no definite colics. The attacks were increasing in frequency.

Examination at the Clinic showed that the liver was definitely enlarged. A diagnosis was made of intermittent biliary obstruction with cholangitis.

Operation was performed May 21, 1920. There was an extensive stricture of the common duct and cirrhosis of the liver. Hepatico-duodenostomy was performed over a small rubber tube. The patient was examined four months later. She had had one attack of colic with jaundice. The X-ray did not show evidence of the tube. The liver was markedly enlarged. Attacks of chills, fever, and jaundice continued.

At operation May 4, 1921, the former anastomosis was found to be contracted and was producing obstruction, and another hepatico-duodenostomy over a tube was performed. A report received one and one-half years later stated that she had had several attacks of colic and jaundice.

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CHRONIC PARTIAL INTESTINAL OBSTRUCTION IN AN ADULT DUE TO ARRESTED DEVELOPMENT OF THE LARGE BOWEL*

By Robert G. LeConte, M.D., Walter Estell Lee, M.D.,
and
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Mrs. A. M. D., age twenty-seven, had suffered four years with manic depressive psychosis, the attacks of acute mania recurring at intervals of about ten months. The family history was entirely negative.

Past Medical History.—The patient had had most of the usual childhood diseases; the history otherwise was negative until the age of fourteen years, when there was an attack of acute appendicitis, which subsided promptly, and an interval appendectomy was done. Complete reports of this illness are not available; the abdomen was opened through a McBurney's incision and its cavity was not explored, but the appendix was said to have been found in the left side. Recovery from this operation was uneventful.

About the age of puberty an enlargement of the thyroid gland developed and persisted, gradually increasing in size. Thyrotoxic symptoms finally became manifest, and the gland was subtotally removed in April, 1922. The pathological diagnosis was toxic adenoma. There was complete relief of thyrotoxæmia, which has not recurred, although the mental condition was not influenced.

Menstrual history—negative. Normal full term labor in April, 1918. There has been no other pregnancy. Child is at present in perfect health.

Present Illness.—Since childhood, and particularly since her appendectomy, the patient has been quite constipated. As the mental disease developed, it was soon noticed that one of the first symptoms of an approaching outburst of mania was the development of extreme and obstinate constipation, movement of the bowels being secured only through the medium of an enema every third day. This constipated condition improved very much as convalescence became established, but never disappeared. It had also been noticed for years that the patient suffered more or less constantly from a pain of obscure origin in the right lumbar region. This was considered to be neurotic, even though it was always worse just preceding a mental outburst, when the constipation was most severe.

After this relation of obstipation to the attacks of acute mania had been observed sufficiently often to give assurance that it was more than a coincidence, gastro-intestinal studies were undertaken. Dr. G. E. Pfahler reported that X-ray examination of the gastro-intestinal tract revealed no abnormalities down to the iliocæcal valve. From this point on, there was an extreme dilatation of the colon, with ptosis and malposition of the hepatic flexure, associated with marked coprostasis. After forty-eight hours, the head of the opaque meal had not reached the rectum; after seventy-two hours a large portion of it was still retained within the colon. The large bowel was then completely filled with opaque enema, which confirmed the diagnosis of malposition and dilatation. Several quarts of fluid were introduced, with no feeling of discomfort or distention on the part of the patient.

Laboratory Examination.—Chemical examination of stomach contents and of the stools was negative. Proctoscopic examination showed that the lower sigmoid was widely dilatable, with an anemic mucosa. Blood chemistry, Wassermann, urinalysis, phenolsulphonethalein tests for kidney function all were within normal limits. Basal metabolism was normal one year after thyroidectomy. Blood-hæmoglobin averaged 90 per cent. Red blood cells from 4,300,000 to 4,500,000. White blood cells on numerous occasions were never increased and the differential count was normal.

* Read before the Philadelphia Academy of Surgery, April 7, 1924.

On the basis of the X-ray findings and the coincidence above noted between increasing constipation and mental outbursts, colectomy was decided upon and was performed October 2, 1923, by Drs. Robert G. LeConte and Walter E. Lee of Philadelphia.

Operation.—The abdomen was opened by a five inch incision through the right rectus muscle. All normal relations were lost. It was some time before the colon could be found. The cæcum was finally located in the true pelvis. It had an unusually long mesentery. Continuous with it was a twisted, looped and distended mass of bowel, all bound together with dense fibrous adhesions, which represented the whole of the normal ascending and transverse portion of the colon, which were absent from their usual position. The omentum was exceedingly fibrous and thickened, and so firmly adherent to the pelvic structures that separation of the adhesions was impossible. It was ligated and cut away and the whole mobile portion of the colon was removed, probably as far as the splenic flexure, or a little beyond. About three-quarters of the length of the large bowel was excised. The end of the ileum was joined to the sigmoid flexure by lateral anastomosis.

Pathological Examination.—The pathological examination of the specimen revealed no gross abnormalities. The epithelium was nowhere atrophic or ulcerated. Microscopically, the only abnormality was an excessive thickness of the muscular layer.

Post-operative Course.—From the surgical standpoint, the convalescence was exceedingly smooth. There was, as anticipated, a marked sedative effect from the operation for a short while, but at the end of ten days it was necessary to return the patient to the mental hospital from which she had come. Except for the sedative effect, the present attack of mania proceeded as usual, uninfluenced by the operation. For the first few post-operative days there were from 15 to 20 liquid stools a day, mostly involuntary. This number has gradually diminished until now there are one or two formed stools a day, entirely under voluntary control. Physically, the patient is much improved. She looks better than she has for years past, her color is healthy, and her complexion clear. She is entirely free of backaches, of which she complained almost constantly before the operation.

An X-ray examination made three months after operation showed the stomach and small intestines normal, with peristalsis normally active. At the end of 24 hours almost all the barium meal had been evacuated. The remaining portion of the colon is from time to time quite rapidly filled by reverse peristalsis, and after a variable period rapidly and completely emptied. An opaque enema distended the rectum, sigmoid, and the remaining pouch of the descending colon, and could be pressed back through the stoma into the ileum. Plates were taken but were not satisfactory.

Discussion.—This case raises several interesting questions. What connection, if any, was there between this pathological condition of the alimentary tract, the hyperthyroidism and the mental illness?

What was the actual state of affairs within the abdomen and what caused it?

The first question is not susceptible of a perfectly satisfactory answer. Sir Arbuthnot Lane,[1] it is true, considers intestinal stasis responsible for whole hosts of pathological conditions, from cold hands and feet and bad breath on, including Graves' disease, and advises colectomy for their relief; but few are willing to go as far as that.

Likewise the relationship between the intestinal stasis and the mental illness must be left an open question. Manic depressive psychosis is almost universally held to be *sui generis;* there is no essential pathology known except a faulty makeup. True, this conception is being questioned at the present day, but is not yet overthrown. Mental troubles do go with megalocolon, but as Hallez [2] says they are usually of the nature of a hypochondriacal depression,

Whether in this particular case there were any relationships between intestinal stasis and mental disease must, I think, be left *sub judice*. No decision will be possible until the lapse of more time.

To the other questions a more definite answer can be given. The condition was evidently not true Hirschsprung's disease, for that is a dilatation of

FIG. 1.—Röntgenogram showing distortion and malposition of colon. The greater part of the colon lies below the level of the iliac crests. Röntgenogram by Dr. G. E. Pfahler, of Philadelphia.

a normally situated colon and it is probably congenital, though urgent symptoms are sometimes late in developing.

Embryology of the Colon.—The fundamental fault in this case was an arrest of development, occurring in early fetal life—probably about the third month of gestation. It will be remembered that the primitive gut early forms a loop directed ventrad, with the vitelline duct at the summit of the loop. The first change that occurs is the formation of the anlage of the cæcum on one side of the caudal limb of the loop. The whole loop then twists about the superior mesenteric artery as an axis, until the cæcum lies under the liver; the entire gut is still connected with a common mesentery. The colon is then pushed out against the parieties by the developing coils of small intestines, until—about the fourth or fifth fetal month—the mesocolon fuses with the parietal peritoneum in the left flank, and across the spinal column and duode-

num, so that the duodenum pierces the transverse mesocolon. Then later, and more slowly, the cæcum elongates, and grows down along the right side of the abdomen, but does not reach its definitive position until several years after birth. In its course, the cæcum and its mesentery also normally fuse with the parieties.

In this long and complicated process of development from the simple fetal to the complex adult relation, an arrest of development is possible at any point. The most common fault is for the cæcum to fail to fuse with the parietal peritoneum, a condition resulting which is known as cæcum mobile. On the other hand, the fusion may progress further than normal and the whole cæcum and even the appendix be retroperitoneal. Very rarely the rotation may take place normally, but the mesocolon fail to fuse—a condition usually called mesenterium commune. Occasionally rotation may not occur at all, or only in part.

Bräunig[3] discusses this subject in a most interesting article, although he gives a slightly different anatomical classification. He considers there are three degrees of failure of the gut to rotate properly. First, cæcum mobile, where rotation has occurred, but the ascending colon has failed to appear. Second, where rotation and fusion have taken place properly from the caudal end of the gut to the splenic flexure, but from there up has not occurred. Third, where there has been complete failure of rotation of the primitive gut, with mesenterium commune.

He quotes a case of the third degree of failure of development, which illustrates the subject very clearly, as ours illustrates the second degree. In Bräunig's patient, the head of the cæcum and the termial ileum pointed cephalad; the continuation of the cæcum coursed into the true pelvis, which was filled with coils of colon, with no small intestines. The colon was attached to a long mesentery, and strangely, was provided with a well developed omentum. The junction of the cæcum and colon was not more than a finger's breadth separated from the junction between the colon and the rectum. Below the stomach was no transverse colon, only coils of small intestines; the greater curvature gave origin to a narrow great omentum, only two finger's breadth wide.

This case of Bräunig's shows complete retention of the fetal condition with entire failure of the primitive gut to rotate. The colon occupies its primordial position, caudad to the rest of the alimentary canal. Our case, on the other hand, is an example of the second degree of failure of rotation, for the relationship of the colon was normal below the splenic flexure. From this point to the cæcum the large bowel was found occupying the true pelvis, its primitive situation below the small intestines.

A curious circumstance, which I am not able to explain, is that both Bräunig's patient and our own had what was apparently a great omentum depending from the colon. The great omentum is entirely a gastric organ, formed from a pouching of the mesogastrium in the process of rotation of the stomach, and only secondarily comes in relation with the transverse colon at a comparatively late stage in development. Evidently, however, it subserves such an important function, whatever it may be, that if there is no great omentum attached to the colon, it is necessary for the organism to improvise one.

The case we report began to show symptoms of chronic constipation

following her appendectomy in childhood. Until that time, the intestinal malformation had caused no symptoms; nor is there any reason *per se* why it should have caused any. Evidently the attack of appendicitis was more acute than was thought at the time; there was probably a certain degree of peritoneal irritation or true peritonitis which resolved spontaneously, but which left behind those crippling adhesions holding the colon firmly kinked and motionless, which was the actual cause of the chronic obstruction.

Summary.—A case is reported of early arrest of development of the alimentary canal. Superimposed on a simple malformation are the sequelæ of a former peritonitis, which kinked and bound the colon together in such a way as to result in chronic obstruction of mild degree. Associated, whether coincidentally or etiologically cannot be determined, is a condition of Graves' disease and of manic depressive psychosis. The embryology of the alimentary canal is discussed in explanation of the condition found.

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HEMORRHAGIC ULCER OF MECKEL'S DIVERTICULUM

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of Madison, Wis.

A careful search of the available literature failing to afford any reference to cases of repeated attacks of severe intestinal hemorrhage due to an ulcer of Meckel's diverticulum, the following case report is presented as worthy of record.

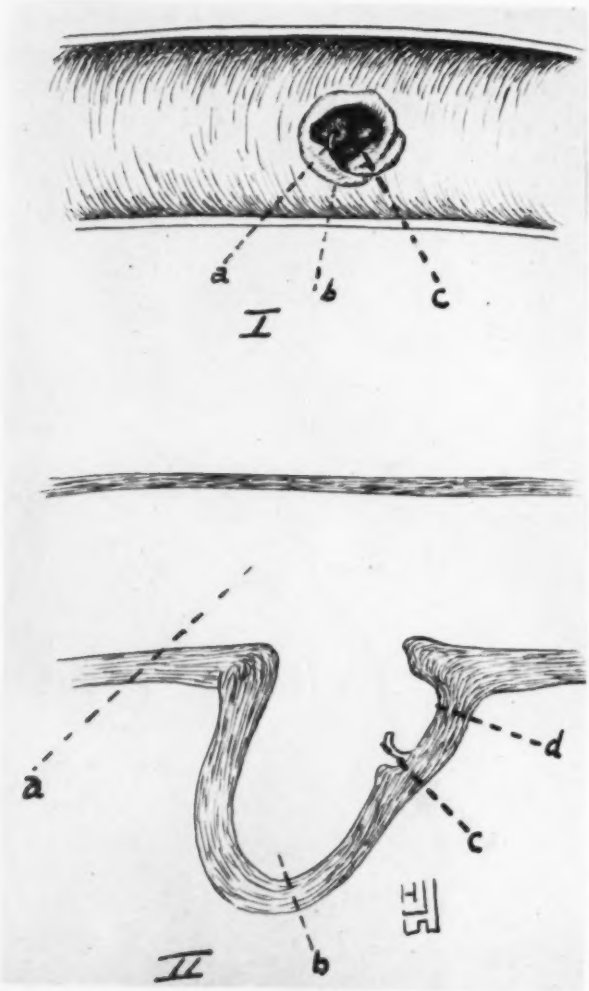

Fig. 1.—Ulcer of Meckel's diverticulum: a. artery; b. margins of opening into diverticulum; c. ulcer.

Fig. 2.—Ulcer of Meckel's diverticulum: a. lumen of bowel; b. the diverticulum; c. artery; d. the base of the ulcer.

W. E., a boy aged ten years, was first brought to the Clinic, March 4, 1912, because of repeated attacks of intestinal hemorrhage. Six months previously he had complained of a rather severe colicky pain in the left mid-abdominal region, accompanied by repeated vomiting at frequent intervals for forty-eight hours. The vomitus had consisted of mucus and bile. There had been no elevation of temperature. The boy had quickly recovered from this attack and apparently had remained well for three months. He then had, at intervals of one week, three quite similar attacks, the last of which was accompanied by severe intestinal hemorrhage. He had not complained of pain after the second day in this last attack nor had he vomited; he continued to pass blood from the bowel for three weeks; the loss of blood had been so severe that his life was despaired of. Since then he had had several prolonged periods of intestinal bleeding unaccompanied by the cramp-like pains of the earlier attacks. During these periods he became extremely anæmic, with marked loss of weight and strength; but in the intervals he gained weight and strength quite rapidly, though the anæmia persisted to a more or less marked degree. The father had taken the child to

numerous consultants and at one time the boy had been under observation for two weeks in a large city hospital where a diagnosis was made of Henoch's purpura with hemorrhages from the intestinal mucosa.

A very appreciable improvement in the boy's condition had recently occurred, the anæmia being much less severe; but the family lived in constant dread of his having another attack with a fatal intestinal hemorrhage. During the few days which the boy stayed in the hospital under our observation, an intensive study of the case failed to lead to even a tentative diagnosis. Physical examination did not reveal any abnormal condition in the chest or abdomen. Proctoscopic examination had been negative as had röntgenograms of the gastro-intestinal tract.

January 14, 1915, the father again brought the boy to this Clinic. The history during the past four years proved to be a repetition of that given on the previous visit. At frequent and infrequent intervals the boy had had repeated attacks of severe intestinal hemorrhage. Some of the attacks had been initiated by cramp-like pain in the left mid-abdominal region. The hemorrhages had often come without warning and at times were so severe and lasting, and resulted in such marked anæmia and asthenia, that the boy had become a chronic invalid.

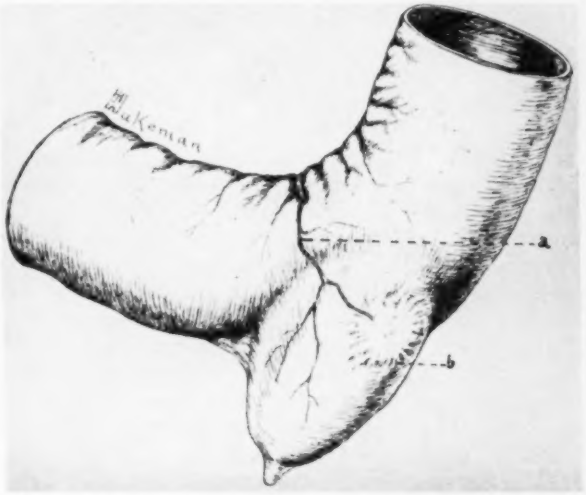

FIG. 3.—Ulcer of Meckel's diverticulum: a. omphalo-mesenteric artery; b. thick scar-tissue at base of ulcer.

The father had given up all hope of a definite positive diagnosis and had returned to the Clinic because the suggestion was given here four years previously that the case might be of a surgical nature and that if the hemorrhages persisted, an exploratory laparotomy, during a period when the boy was in fair condition, would be warranted. This opinion had been based upon the facts that:

1. A positive diagnosis of any true blood dyscrasia could not be demonstrated.

2. The attacks were often initiated by severe cramp-like pains definitely located in the left mid-abdominal region.

3. In the occasional prolonged intervals between the attacks the boy gained rapidly in every way and the blood picture became practically normal.

Although the patient was again thoroughly examined, we failed to find any evidence on which to base a diagnosis, beyond that of a probable surgical condition.

Operation.—Operation was performed under ether anæsthesia. A 10 cm. left mid-rectus incision was made to permit a thorough examination of the abdominal contents. The peritoneum was normal. Palpation and inspection of the rectum, rectosigmoid, ascending, transverse and descending colon failed to reveal any abnormal condition. The stomach and duodenum were normal, as were the liver and spleen. A great many palpable lymph-glands were found in the mesentery, the largest of these measured 1 x 1 cm. and some of them were calcareous. Beginning at the ileocæcal junction, the small intestine was stripped between the fingers. A conical, indurated mass 6 x 3 cm. was found protruding from the ileum 50 cm. from the ileocæcal valve; the base of the outgrowth was incorporated in the tissues forming the wall of the ileum in the manner characteristic of a Meckel's diverticulum. This Meckel's diverticulum was so indurated and chronically

inflamed that it was not possible to excise it and leave a patent condition of the bowel. The remainder of the small intestine was in all respects normal. A segment of ileum, 12 cm. in length with the diverticulum, was resected and a lateral anastomosis made with careful closure of the mesenteric gap.

Gross Examination of the Specimen.—The diverticulum was of the stumpy, conical type without any terminal ligament. On palpation, the wall was so indurated that it required considerable pressure to indent it. The maximum thickening was at the base of the distal side and continued on into the wall of the ileum for about one-half of its circumference in such a way as to diminish the lumen of the bowel to a considerable extent. A 20 degree angulation of the bowel on its mesenteric side was noted. During exacerbations of the chronic inflammatory process there would undoubtedly be sufficient encroachment on the bowel lumen to give rise to partial intestinal obstruction. By dividing the wall of the bowel opposite the diverticulum a very excellent view was afforded of the exciting factor of the patient's repeated intestinal hemorrhages. The walls of the diverticulum at its base were from 1 to 2 cm. in thickness and an indurated crater ulcer 1 cm. in diameter was situated in the thickest part of the wall of the diverticulum, partly in the margin where the osteum of the diverticulum begins, but four-fifths of it lying within the diverticulum itself. In appearance it resembled an indurated duodenal ulcer. The base of the ulcer was entirely bare of mucosa and at the margin of this area an eroded vessel, which looked like an artery, projected from the surrounding surface in an erectile manner. This particular vessel had undoubtedly been the source of the many hemorrhages. It was apparently a terminal branch of the omphalo-mesenteric artery which was found coursing on the surface of the diverticulum.

Subsequent History.—The boy made an uneventful recovery and has remained well. Unfortunately the specimen, which was sent to the pathologic laboratory of the University of Wisconsin, was mislaid and efforts to find it have thus far been unsuccessful. The presence of numerous enlarged lymph-glands in the mesentery of the small intestine, some of these glands being decidedly calcareous, would suggest that the ulcer might have been tuberculous in nature.

A METHOD OF HERNIOTOMY UTILIZING ONLY WHITE FASCIA

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There has been a steadily increasing recognition of the fact that our methods of curing inguinal hernia are far from satisfactory. The very fact that the literature is constantly full of new operations or modifications of old ones is suggestive. A study of the recurrence rate amply confirms our fears.

Prior to Bassini,[1, 2] Wood,[3] in 1886, reported 27 per cent. recurrences, and Bull,[4] in 1890, 36 per cent. Both were ready to admit defeat and call hernia surgery a failure. Bassini's achievement of only 2.8 per cent. recurrences[2] gave surgeons a very exaggerated idea of the efficacy of his method. These concepts prevailed for about 20 years and we hear Murray[5] claim 1.7 per cent. recurrences. Judd,[6] 2.5 per cent., Bull and Coley[7] 0.8 per cent., all just prior to 1910. We know now that such statistics meant nothing because they were not arrived at by proper follow-up methods. Since then the impression has grown that all was not well and the publication of series of actually traced cases have changed our optimism to pessimism. The following three series, from the Massachusetts General Hospital, Bellevue in New York and Johns Hopkins respectively, are representative of results in America. Similar figures are available in the foreign literature.

Davis[8]	9 per cent.
Erdman[9]	7.5 per cent.
Taylor[10]	10.9 per cent.

(The following references in the bibliography contain a series of traced cases and if consulted will dispel all doubt that the above figures have a very general application.)

Davis,[8] Erdman,[9] Taylor,[10] Schwartz,[11] Ricketts,[12] Galeazzi,[13] Oudarde and Jean,[14] Russell,[51] Lameris,[16, 17] Masson,[18] Salzer,[19] Hoffman,[20] Murray,[5] Murphy,[21] Fantine,[22] Bassini,[2] Harrison,[23] Downes,[24] Bloodgood,[25] Esten.[26]

This growing dissatisfaction with the results of our work is shared by almost all students of the subject except one—perhaps our greatest—Coley. His yearly reviews in "Progressive Medicine"[27] reveal a complacency and decided feeling of satisfaction with the Bassini type of operation. However, allowing for his enormous experience, which none of us can ever hope to equal (nearly 10,000 cases in 1924),[28] it is only reasonable to call attention to the great preponderance of children in his material and the corresponding scarcity of large hernias and direct ones. His own estimate of his recurrence rate, 2 per cent.–3 per cent. is not based on follow-up reports and one cannot doubt that it would correspond to a far higher one in more difficult cases. For recurrences in children, see Kovacs,[29] one recurrence in 144 traced cases; Salzer[19] none in 50 traced cases; Stiles,[30] 360 cases, 4 recurrences. Sultan,[31]

Ochsner,[32] and many others have noted a low recurrence rate in children. Murphy,[21] in a very elaborate analysis, reports a gradually increasing rate from 1 per cent. under one year to 9 per cent. in patients over 60.

All the above material represents the results in the foremost clinics and for the most part omits strangulated hernias and unusual ones and hence it is reasonable to suppose that less experienced operators have still worse results. We must face the fact that herniotomy is one of the operations most commonly done by the beginner in surgery.

A more detailed study of the causes of recurrence [8, 9, 10] reveals the significant fact that these failures cannot be passed off as unavoidable infections. To be sure sepsis accounts for many, but in no clinics do infection occur in any such numbers and itemized reports leave no doubt that the majority of failures can only be attributed to faulty planning of our operations. Any contemplated change in our methods must consider two factors, one very old and the other rather recently brought to light.

(1) *The Endo-abdominal Fascia.*—So many names have been applied to this unit structure that the utmost confusion reigns as to just what it is. Not only have sage anatomists changed its name for every region of the belly but even in the inguinal region no agreement seems to exist. Be it called endo-abdominal, transversalis, verticalis inguinal, iliac or what-not, it is all the same to the surgeon, who in every other laparotomy would never dream of omitting to close it. A hundred years ago when such surgeon-anatomists as Cooper [33] and Scarpa [34] made beautiful dissections of hernias they understood clearly that one of the most important elements in the pathology of hernia was the giving away of this fascial envelope of the belly. In some cases the hole for the exit of the cord was unduly large. In others the portion next to Poupart's ligament had simply been thinned out and stretched. Much discussion occurred as to whether this was a cause or effect of hernia. Our present knowledge of the wholly congenital (saccular) origin of the disease (Coley *et al.*[35] and Russell [36]) leads one to suspect that this deficiency is also congenital. Operative findings in children also tend to support this view. Mere calling attention to the fact that at this point strangulation nearly always occurs, is ample proof that this is the real internal ring and that the abdominal muscles have nothing whatsoever to do with the internal ring.

Older surgeons, such as MacEwen,[37] of Glasgow, and Marcy,[38] of Boston, planned their operations with this structure clearly in mind. Then, when in 1889, Bassini made Padua the Mecca of all herniotomists, the attention of every one was rivetted on the conjoined tendon as the real structure to be used in the closure. That so many writers forgot his teaching was no fault of his, except in so far as he relapsed into the deepest silence. A surgeon, E. Wyllys Andrews,[39] who visited his clinic and described his exact technic (" The Bassini operation as done by Bassini himself ") spoke as follows:

" Now the dissection of the peritoneum away from the internal ring * * * has also had the effect of loosening the transversalis fascia and internal

oblique muscle * * *. These as every one knows are the structures to be included in the Bassini operation in the first or deep line of suture, intended to restore the enlarged ring to its normal size. To facilitate the insertion of these stitches a forceps is made to grasp the transversalis and internal oblique, pointing from the internal ring inward, one blade just outside the peritoneum, *i.e.*, the blades grasp the whole of the posterior wall except the peritoneum."

Bouveret,[40] a contemporary, in describing Bassini's operation, published

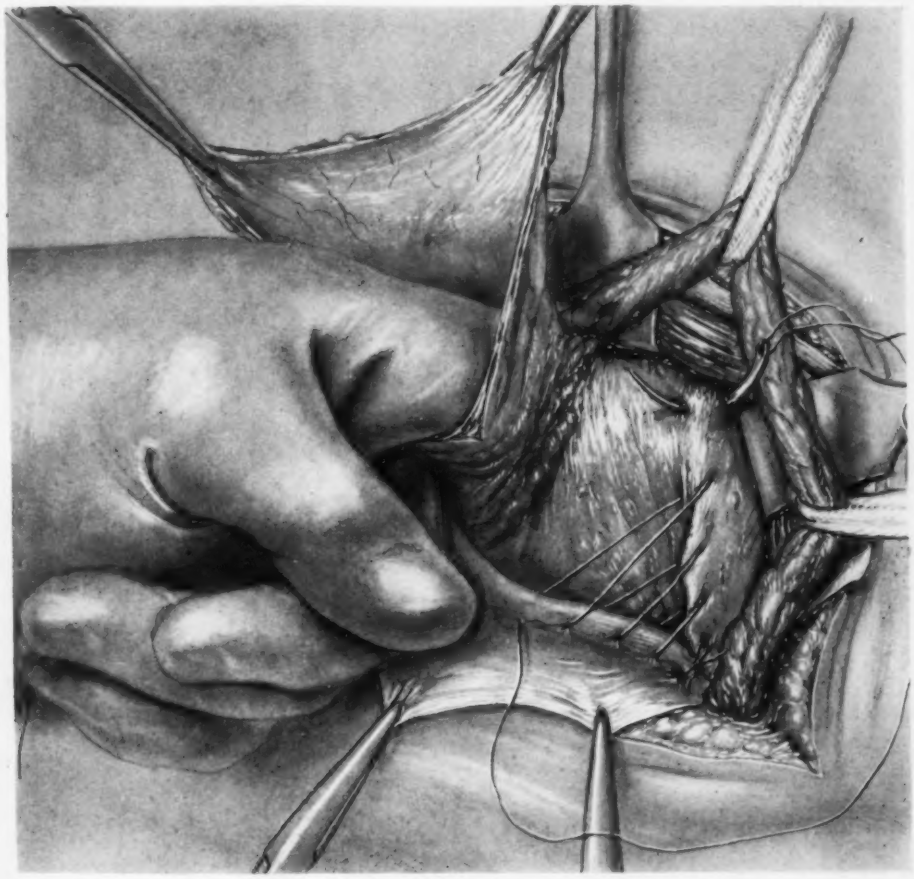

FIG. 1.—Finger as a guide to outline deep ring.

this diagram showing clearly the recognition of the necessity of closing the hole in the endo-abdominal fascia. (Fig. 1.)

It goes without saying that in the vast majority of clinics the Bassini operation is most emphatically not done in this manner any longer, although recently Pitzman,[41] LaRoque,[42] Druener [43] and others have described methods, some admirably designed to correct this defect in our technic. Even now it is clearly recognized that the most efficient way to close a hernial ring is from within. If one is performing any other laparotomy in the neighborhood, a few

stitches in the peritoneum and fascia are far more effectual than any other type of herniotomy. One has the feeling that he is really sewing up a hole and not going through the motions of a complicated plastic operation. The question remains, however, even if the Bassini operation were properly done, is it anatomically and physiologically correct?

(2) Use of Red Muscle a Mistake.—It is a commonplace that in a Bassini operation our inner row of stitches are in red muscle, not in tendon. The conjoined tendon as seen in the dead house simply does not exist in men with hernia in most cases. Bloodgood,[44] Blake,[45] Hessert,[46] and many others have emphasized this fact. Polya[47] showed clearly that the lower fibres inserting onto the pubis are absent in hernia patients and the insertion of the lowest fibres was onto the lateral edge of the rectus sheath, averaging 2-3 cm. above the bone. He measured 100 consecutive cases of hernia with the following results:

Polya.—Length of rectus bordering inguinal canal.

Cases	Cm.
1	0
7	1
31	2
28	3
17	4
9	5
5	6
2	7

Ferguson[48] demonstrated that a similar condition existed at the lateral end of the canal and that in men with hernias the origin of the internal oblique was farther out on Poupart's ligament. It can then be definitely assumed that the red muscle bordering the canal lies at a considerable distance from the inguinal ligament and can therefore only be approximated to it under considerable tension, especially near the pubic spine. The doubt at once arises, can it be made to adhere to Poupart's?

Oudarde and Jean[49] studied 183 recurrent hernias operated upon in the French Navy. In practically every case the red muscles had pulled away from the inguinal ligament. Moschcowitz,[50] Polya,[51] Walker,[52] Harrison[53] and Lusk[54] have published similar observations and all surgeons who have operated upon many recurrent hernias will vouch for the truth of such statements. Until recently this separation of red muscle from Poupart's was thought to be the cause of the recurrence. We are now being compelled to face the fact that such a union never occurs. The most that can be hoped for is the creation of a thin layer of scar tissue between these structures.

McNealy[55] has observed several old herniotomy wounds at post-mortem. He finds that the red muscle pulls away in all cases even though no new hernia occurs. I have had the opportunity to make one similar observation ante-mortem. This occurred in a young man who had been operated upon for cryptorchidism about one year before. There was no recurrence of the

hernia but the testicle had retracted into the canal again. I found that, although the posterior wall of the canal felt firm and resistant, the margin of the internal oblique was nowhere in contact with Poupart's ligament where it had been stitched by the previous operator.

Seelig and Chouke [56] attacked this problem experimentally in a very clever manner. By making a curved incision in the fascia lata of a dog and reflecting the flap, a sharp edge can be produced similar to that of the inguinal ligament. They sutured this to the underlying muscles and found that in every case the stitches promptly cut through and no adhesion was created. Silk suture lasted only a few days longer than catgut. The only agent capable of producing real adhesion was extensive infection, an agent whose aid surgeons would scarcely solicit. They also called attention to the fact that such a union had no parallel in other normal structures and that theoretically one would not expect it to occur. Dowd [57] seems to have had a similar thought in 1914 and Druener [43] in 1919.

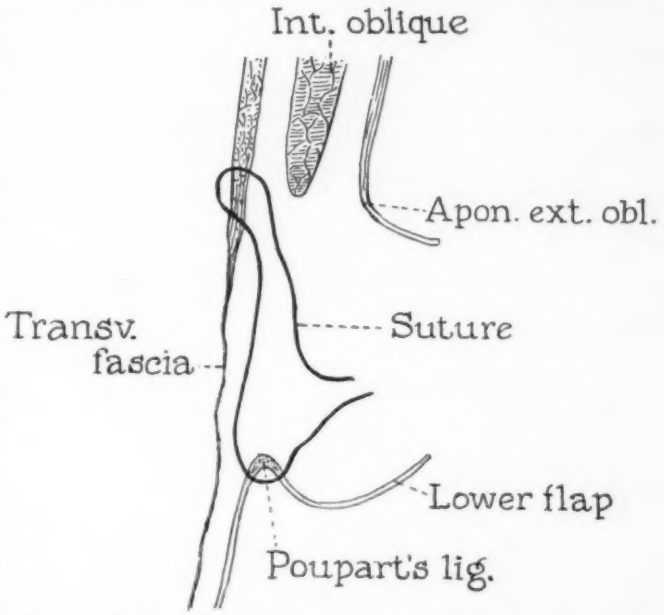

FIG. 2.—Sagital section of layers showing deep suture placed beyond thin edge, thus obtaining a strong layer.

Not only theoretical and experimental observations are available as proof of this point. Clinical experience of many surgeons may be added. Hull,[58] Hoffman,[20] Salzer,[19] Pitzman,[41] and Lameris [17] have had the courage to abandon the red muscle suture and the results have amply warranted their daring. There are now on record over 2000 herniotomies consisting solely of sac removal which were followed up and reëxamined. The recurrence rate is slightly less than that of comparable series done by standard methods. Furthermore, it is now being done as a routine in many European clinics and further information will soon be available.

It is not enough to demonstrate the uselessness of internal oblique suture. On that basis alone conservative surgeons may argue that it might conceivably be of value and should therefore be retained. Can we say that it is positively harmful? First let us consider the function of this muscle. In a normal man the lowest fibres have a definite sphincter action. They arise from Poupart's

and insert just beneath it on the pubis. Contraction of these fibres will approximate them to the ligament. This has been compared to the shutter action of a camera by Keith.[59] It is strictly comparable to the sphincter ani externus, except that it represents only one-half of it, the other half being a solid structure. In neither case are circular fibres necessary to accomplish sphincter action. The cremasteric fibres represent the internal circular sphincter. The weakness and inefficiency in man is evident but it is interesting to note how they hypertrophy under the stimulus of attempting to retain a long standing hernia.

In nearly all animals except man the patent tunica vaginalis persists throughout life but inguinal hernia is rare because of this sphincter action. In rodents the testes pass freely back and forth from the abdomen to the scrotum in response to physiological conditions but even this constant distention of the canal is counteracted by the sphincter action of the cremaster. This is called the cremaster externus in animals and is a well developed circular set of fibres. Even the larger herbivora with the enormous intra-abdominal pressures common on such diets (hay-belly) are thus able to protect wide open hernial sacs.

In discussions of the etiology of hernia in man one often hears that the upright position is to blame. To this ruinous primate blunder has been attributed every bodily ill from colonic stasis to flatfoot but I doubt if any wilder absurdity has been perpetrated than to ascribe to biped locomotion a disease occurring most commonly in infancy.

Of course any modern speculation on the etiology of hernia must include the preformed sac. But anatomical records point unmistakably to the fact that most men with preformed sacs never get hernias. About 35 per cent. of three months old infants have potential hernia sacs, (Murphy);[21] in middle age they are found in 23 per cent. of cadavers (Eccles);[60] and Keith[59] found 12 per cent. in the very aged. In the latter figures inguinal sacs are in a majority, umbilical and femoral ones predominating. Compare these figures with the incidence of hernia. About one in sixteen born will acquire hernia (Berger).[61] Why do not all these potentially ruptured become actually so? The answer is the same as in the case of the lower animals. We have our cremaster as one guard and the internal oblique as a second. The first is admittedly weak. Consider now an internal oblique with the lower fibres absent as noted above. (Table II.) It has only one end attached to poupart's, the outer. The inner end is 2-3 cm. away on the rectus sheath. Its efficiency as a sphincter is seriously impaired. The condition is again parallel to a sphincter ani. If during childbirth the anterior ends of the rectal sphincter are separated from each other, its mechanism is ruined. Repair consists simply in bringing them together again. No actual destruction of muscle has occurred. This anal sphincter consists simply of the medial fibres of a much larger muscle, the levator ani. So the inguinal sphincter consists of the lower fibres of the internal oblique.

The lowest fibres are the ones that count and their destruction by ill

advised suturing will utterly wreck the inguinal sphincter. Whether such sutures cut off the nerve filaments to the lowest fibres cannot be positively stated but the studies of Dowd [62] and Moschcowitz [63] leave considerable doubt on this score. What other explanation can be offered for the fact that about 4 out of 7 recurrences in oblique hernia are not oblique but direct and come

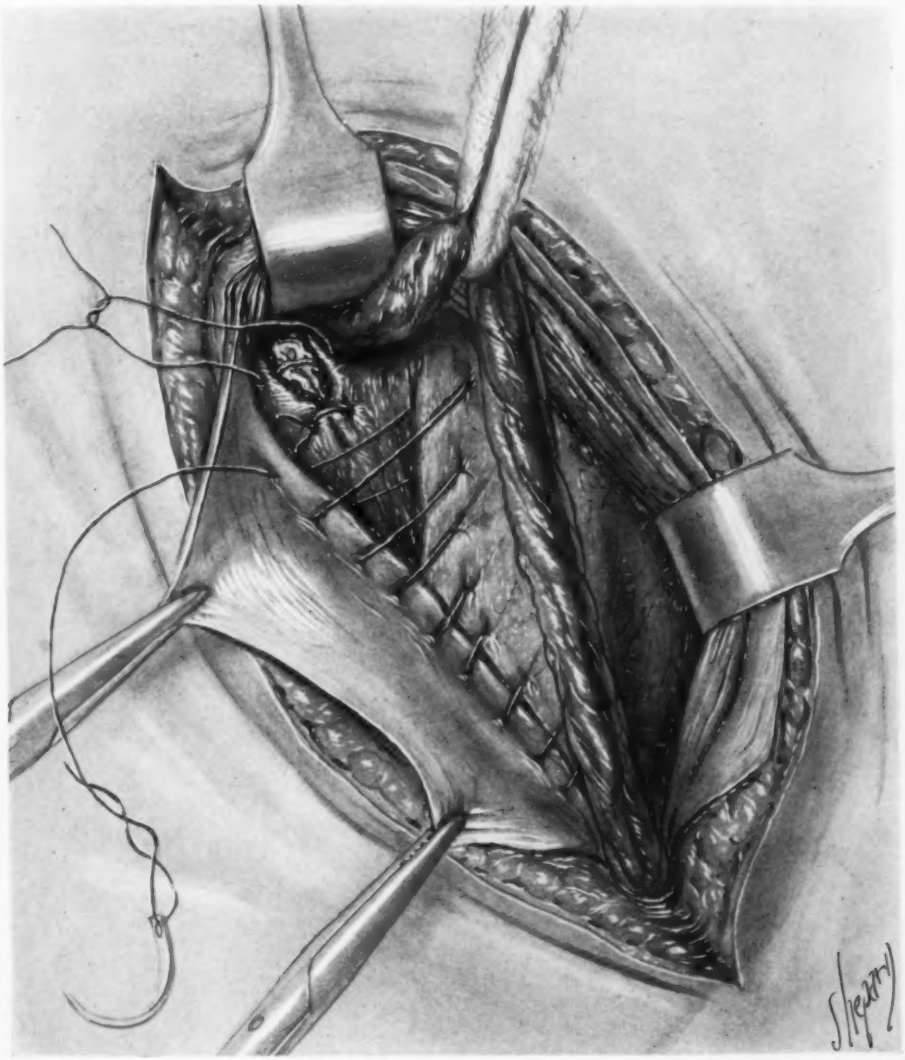

FIG. 3.—Transversalis fascia instead of red muscle in deep suture.

out just above the pubic spine? (Burian,[64] Fantino,[22] Masson,[18] Erdman.[9]) The only possible interpretation is that in these cases our stitches in the lower end of the canal have not only failed to strengthen it but instead have brought about hernia formation where none existed before.

As has been amply demonstrated, simple sac removal will cure most hernias.

The results are on a par with those attained by older methods but the recurrence rate is still far too high. The 3 per cent. reported by Lameris [17] by this method excluded all direct, all strangulated and unusual hernias. If these were included the rate would undoubtedly be far higher. The only really important advance in hernia surgery since 1889 is the principle of overlapping

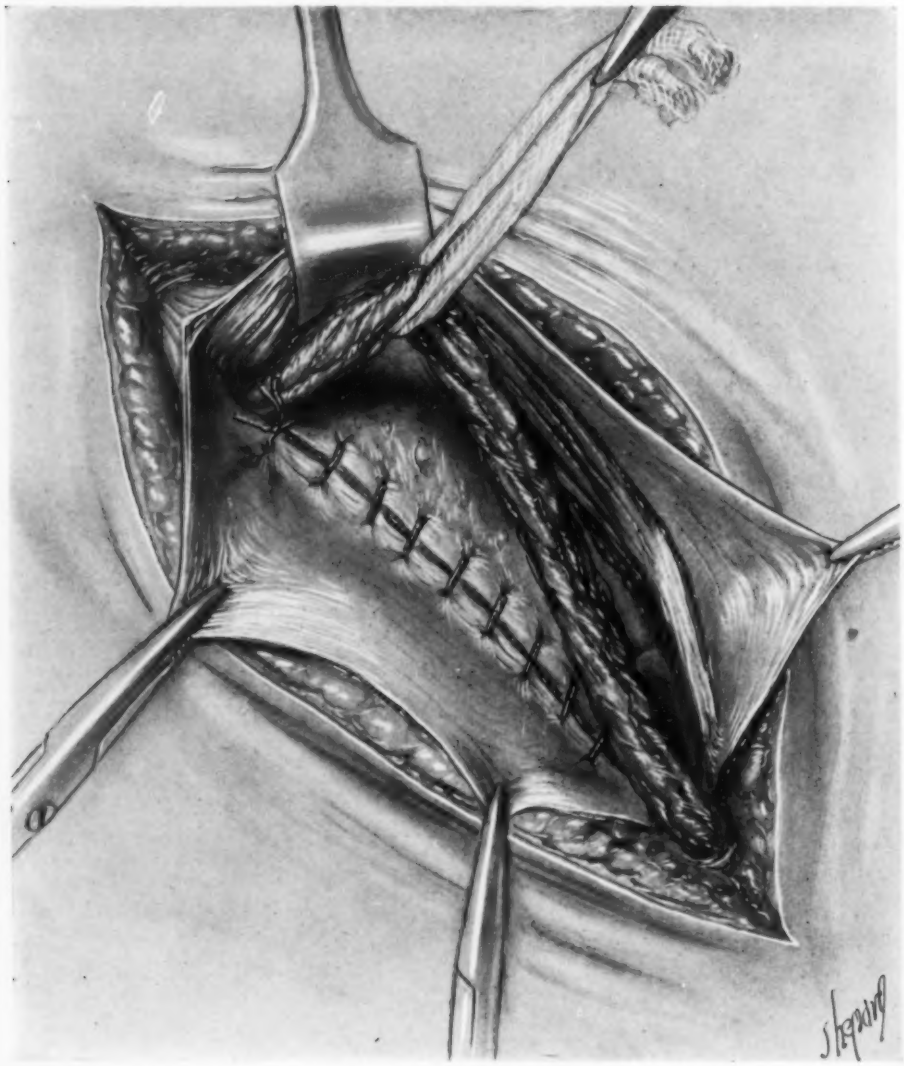

FIG. 4.—Deep layers. White fascia sewn to Poupart's, avoiding red muscle.

the fragments of the external oblique aponeurosis published by E. Wyllys Andrews in 1895 [65] and again in 1905.[66] It was devised independently in Switzerland by Girard [67] in 1900 and often goes under his name in Europe. This principle lends itself admirably to adoption in a modified form as a

reinforcement for the posterior wall of the canal after full utilization of the endo-abdominal fascia.

Operation.—The operation I have used to meet these conditions utilizes only white fascia to repair the canal, in contradistinction to the use of red muscle.

The canal is laid open in the usual manner and the cord lifted with all its

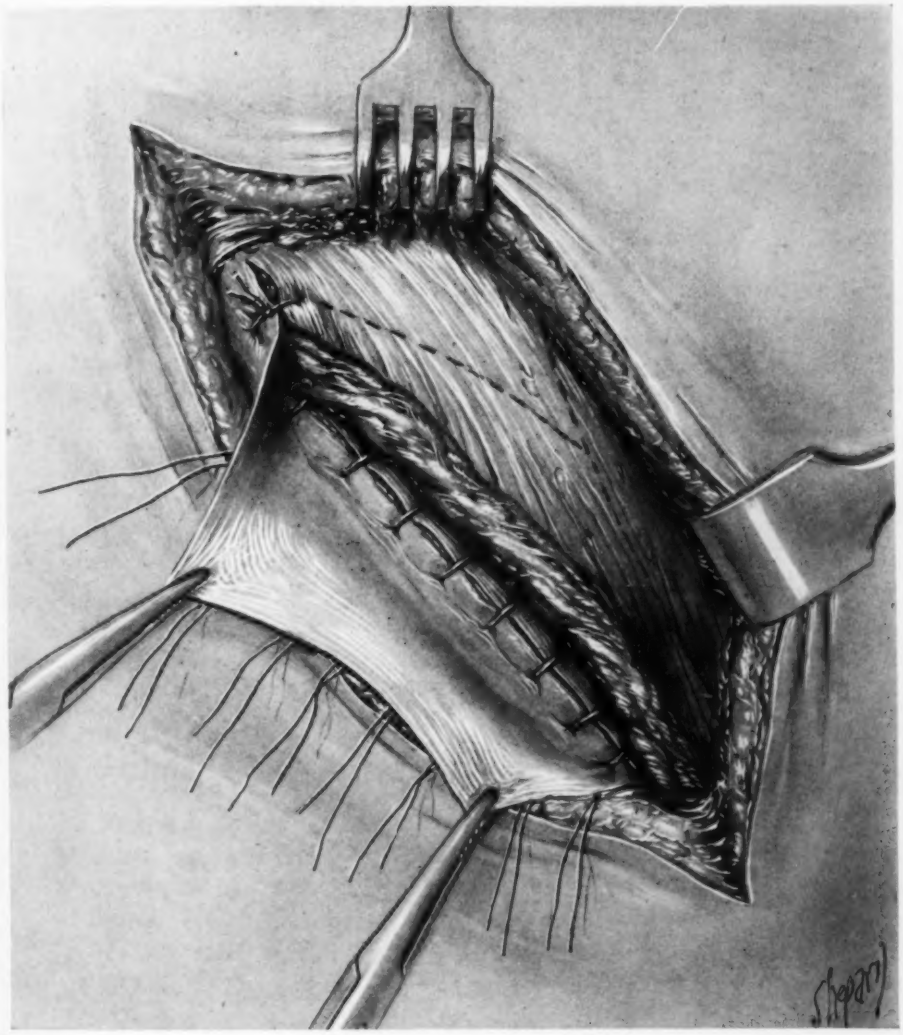

FIG. 5.—Additional layer (external oblique aponeurosis) imbricated behind cord.

coverings. This point is important as it is necessary to clear the floor of the canal of all impedimenta. The cord is then opened, the sac isolated and emptied. The floor of the canal is then cleared of all fat and areolar tissue and the deep epigastric vessels identified and freed so that they may not be injured. The index finger is now inserted into the belly underneath the canal

to act as a guide to our stitching. With this as a safeguard we are able to safely place deep stitches in the deep layers of the abdominal wall. (Fig. 1.) The red muscles are now retracted exposing the endo-abdominal fascia. The type of fascia can now be determined. If the fascia is strong but simply has too large a hole in it our work is easier. By a continuous stitch beginning near the pubis this layer is tightened and the orifice narrowed to the proper dimensions. In the vast majority of cases this fascia, particularly in the part close to Poupart's, is too lax and thin to be of any value. This thinning, however, is always quite local and in these cases *one never has to go more than 2–3 cm. away from Poupart's to find material of ample strength.* (Fig. 2.) This obviously cannot be sutured to its weak inguinal portion but can very readily be approximated to Poupart's from the pubic spine to the internal ring. I find that this layer is extremely loose, elastic and movable and in every case have been able to pull it down to Poupart's with a minimum of tension. The suture is best begun near the pubic spine and is continuous right up to the internal ring. The needle first picks up a liberal bite of the endo-abdominal fascia. It is pushed through directly onto the guiding finger, brought out again and then picks up the edge of Poupart's from without inward.

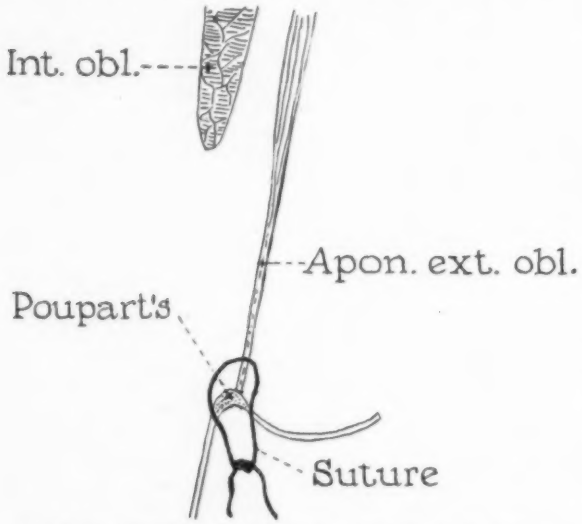

FIG. 6.—Diagram showing knots of deep suture outside fascia.

When this manœuvre has been completed, three clamps are placed on the edges of the internal ring which is now a clear anatomical entity. (Fig. 3.) Before in many cases it could not even be recognized. The sac is now transfixed, ligated and excised and another stitch or two taken in the endo-abdominal fascia over the stump leaving only a very small hole for the cord. (Fig. 4.)

For reasons above stated the red muscles are ignored.

The upper flap of the external oblique aponeurosis is now sewed to Poupart's ligament with a row of interrupted sutures. (Fig. 5.) These are applied in such a manner that the knots are all lying outside the canal. Each stitch begins in Scarpa's triangle, goes under the ligament, emerges in the canal, gets a bite of the aponeurosis and then goes out through the lower fragment of the aponeurosis close to Poupart's (Fig. 6). I usually put one or two of these stitches above the cord so as to continue the overlapping of the external oblique a little lateral to the canal.

The cord is then replaced in its newly formed bed and the lower flap of aponeurosis sutured over it as a roof for the canal. (Fig. 6.)

I use only kangaroo tendon in the closure. As Marcy [68] demonstrated over fifty years ago (1871), this substance causes less irritation in the tissues, is easier to sterilize with less chemical action and the experience of many

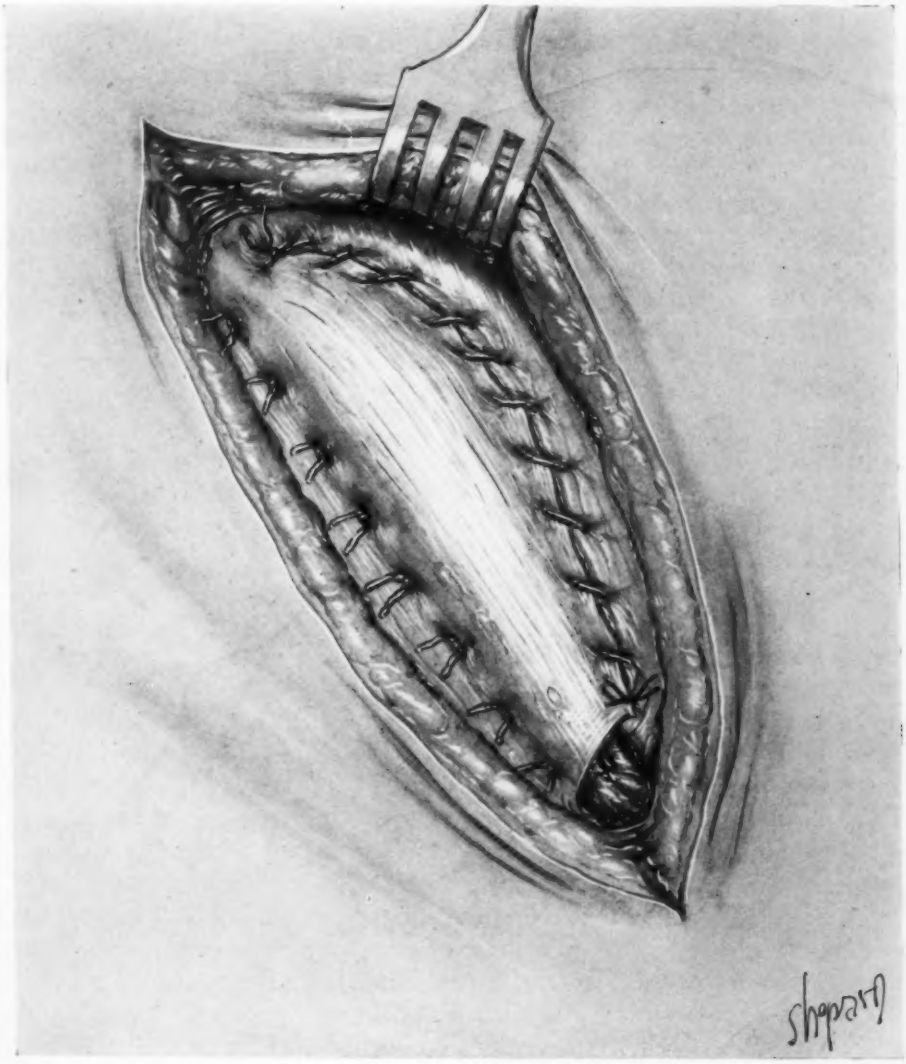

FIG. 7.—Last step of Wyllys Andrews' imbrication suture.

surgeons has amply proved that in practice it is productive of less stitch infection than any other suture material. The only objection to it is the large knots required and this is at least in part overcome by leaving the knots outside the canal.

Discussion.—While the use of the endo-abdominal fascia has been advo-

cated by a considerable number of surgeons especially in recent years, it has usually been dismissed by most with the statement that it is too weak and thin to be of any value. I believe that these failures have been due to the failure to recognize that this deficiency is entirely local. If one takes the average case and pushes ones finger straight down into the abdomen in the lower inguinal region little resistance is encountered. However, if one goes a little distance upward a strong membrane is always encountered. One must remember that this fascia is not normally attached to Poupart's at all, but to the bone under it and therefore it often cannot be found in the space between.

Incidentally my experience has been diametrically opposite from Pitzman's.[41] He says that in the presence of large hernias this layer hypertrophies and it is weakest in the normal. My own observations have been that it is strongest in smaller hernias and is most attenuated in the older larger ones. Of course the sac itself often becomes very thick in large hernias but even in them it is mainly the distal part of the sac and the neck tends to remain thin.

In many cases under the guidance of the finger in the abdomen it is possible to place a row of hæmostats on the endo-abdominal fascia beneath the margins of the retracted muscles and feel it being lifted away from the peritoneum. In such cases the finger safeguard may be dispensed with and our stitches inserted more rapidly and easily. In other cases the fascia and peritoneum are too closely related and the stitches must enter the abdomen protected by the finger.

Since 1918 I have used this method occasionally in small hernias. Recently, however, it has been applied in several large and very difficult ones and the results were so pleasing that it has been adopted as a routine. The inner row of stitches alone appears ample to cure the rupture and if the patient strains or coughs at this point in the operation no bulging will be noted in the floor of the canal. This is true even in the direct type. In direct hernias if a little traction on the cord be made a small oblique sac will always appear. This is probably an artifact in many cases but it serves just as usefully as a point of entrance for the guiding finger. Previous to the adoption of this method I had usually ignored the small dome-shaped sacs in direct hernias, because I was afraid of injuring underlying structures with my needle. I have been quite surprised to find that even the very thin fascia over such sacs becomes quite strong a few cm. away from Poupart's and permits a solid closing of the gap. In all direct hernias I make it an invariable rule to use the rectus sheath in the closure as well as the external oblique aponeurosis. This is best accomplished by the Halsted [69] technic.

This type of operation may not seem to lend itself so well to the use of local anæsthesia but if for any reason a general anæsthesia is contra-indicated the fascia and peritoneum of the anterior abdominal wall can be blocked above the point of suture underneath the retracted muscles. On the side next to the ligament this infiltration is much more difficult and on account of the fear of injury to the great vessels may not be thoroughly carried out.

SUMMARY

1. The importance of the endo-abdominal fascia is emphasized.

2. Considerable evidence is presented that the use of the internal oblique is not only futile, but also probably actually harmful.

3. An operation is described in which the restoration of the canal is made using only white fascia. The floor consists of the endo-abdominal fascia plus the external oblique. The roof of the left-over lower fragment of the external oblique aponeurosis is overlaped or imbricated in front of the cord.

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THE JACK-KNIFE POSITION FOR PATIENTS AFTER OPERATIONS FOR ABDOMINAL HERNIA

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As a means of lessening recurrence, the value of the jack-knife position is not generally appreciated. Much time and skill may be spent in carrying out the minutest steps in technic in a complicated operation, but the position of the patient during the tying of the deep sutures is usually incorrect, and his position in bed after the operation is too often left to the nurse, or to the patient himself, who assumes a position he finds most comfortable.

In the operation for inguinal hernia the best exposure of the field of operation is obtained by keeping the thigh extended until the deep sutures are ready to be tied, when it should be elevated, adducted and rotated inward. This reduces the distance between Poupart's ligament, the internal oblique and conjoined tendon from 25 to 50 per cent., depending on the size of the opening and the development of the muscles and fascia; and the sutures can be tied without tension, even in large hernias.

If the operating table cannot be adjusted so as to elevate the patient's shoulders and knees, the jack-knife position can be secured by the use of pillows, or by placing the patient's foot in a leg holder elevated to the proper position. This relaxed position should be maintained while the patient is transferred from the operating table to his bed.

When the fasciæ and muscles are brought together under tension, there is a natural tendency for the sutures to stretch and the knots to tighten, and as a result the stitches cut through the tissues which also tend to separate parallel with their fibres; consequently the approximated wound edges are not in firm contact and there is often a gap in the interval midway between the sutures.

The patient in bed should have his shoulders and knees elevated 25 to 45 degrees. If this posture cannot be secured by adjustments on the bed, a back rest can be used to raise the shoulders and a pair of pillows placed under the knees of the affected side. (Fig. 1.) The patient is usually more comfortable if both knees are elevated.

The jack-knife position should be maintained for five to ten days, depending on the size of the hernial opening and the tension on the sutures at the time of operation.

The patient is kept in bed ten days to three weeks, depending on the variety of the hernia and the size of the opening. Aged patients are gotten out of bed as soon as possible, and all patients should be cautioned not to do any work for three months after the operation, and only light work for the following six months. They should be warned against a gain in weight,

FIG. 1.—Desirable position after operations for abdominal hernia.

which increases intra-abdominal tension and puts added strain on the wound, thus favoring recurrence. A change of work, which brings new groups of muscles into use thus lessening the chance of recurrence, should be advised.

CONCLUSIONS

The jack-knife position—elevation of the shoulders and knees—reduces the hernial opening 25 to 50 per cent.; facilitates the tying of the deep sutures; takes the strain off the stitches during the process of repair; gives a broad firm union and reduces the percentage of recurrences. This position should be maintained for five to ten days after operation and is usually to be advised for all patients following operation for inguinal, femoral, umbilical and ventral hernia.

THREE THOUSAND CONSECUTIVE HERNIOTOMIES*

WITH SPECIAL REFERENCE TO RECURRENCE, BASED ON EIGHT HUNDRED AND THIRTY SEVEN FOLLOWED CASES

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ASSISTANT SURGEON TO THE HOSPITAL FOR RUPTURED AND CRIPPLED

During the period from August, 1919 to May, 1923, there have been performed at the New York Hospital for Ruptured and Crippled, three thousand operations for hernia. In the period from 1891 to 1919, there were performed seven thousand operations. Thus we have a series of ten thousand consecutive herniotomies performed in an institution where methods of operating, after-care, and follow-up, make statistical study of interest and value. Since a previous communication by Coley (William B.) and Hoguet [1] has dealt with the first series of seven thousand cases, it remains to discuss the later group which differs from the former by reason of the larger proportion of adults, due to the opening of the male adult ward in 1916. Any consideration of results in operations performed for hernia should take into account the age of the patients, and the types of hernia presented. It is of little value to lump all cases together and quote percentage-recurrence based on the entire series. Therefore, each group will be discussed separately. For the purpose of classification, all cases fifteen years of age or over, are considered adult.

Indirect Inguinal Hernia.—1. *In Male Child.*—There were 1013 operations in this group (263 follow-up cases) with no recurrence. This contrasts somewhat favorably with the results in the first 7000 cases where, in 4807 operations, there were 19 recurrences or a percentage of 0.39. We believe that in children, recurrence after operation for indirect inguinal hernia should be reducible practically to zero. The improvement in our later series may be attributable to a better standardization of the Bassini operation which has been the routine operative procedure for this group.

2. *In Male Adult.*—In this class there were 1155 operations, or more than twice as many as had been performed in the first 7000 cases. There were 28 recurrences in 332 followed cases, or 8.7 per cent. All degrees of indirect hernia from bubonoceles to large scrotal and sliding herniæ are here included. The typical Bassini operation was used in the great majority of cases; but with a suture above the cord to limit the size of the internal ring.

3. *In the Female Child.*—Here were found 124 operations with no recurrence, in the 22 followed cases, which is according to expectation as stated above.

4. *In Female Adult.*—There were 113 operations, of which 23 were followed, with one recurrence, or 4.4 per cent. The lower incidence of recur-

* Read before the New York and New England Association of Railway Surgeons, November, 1923.

rence in the female is due, no doubt, in part to the fact that less strain of an occupational nature is thrown upon the repair, but more important is the completeness with which the canal may be closed in the female, as there is no cord to be considered. The operative procedure differs from that employed in the male merely in the fact that the round ligament is not transplanted.

Direct Inguinal Hernia.—1. *In Male Child.*—Four cases only were operated upon (only 1 of which was followed) with no recurrence. This type of hernia is very rare in children. The usual Bassini technic has sufficed to cure the 24 cases that have been observed in this hospital in the entire 32-year period.

2. *In Male Adult.*—Of the 280 herniotomies for direct hernia in male adults, 14 recurred, or 16.4 per cent. of the 85 followed cases. This constitutes the most troublesome group of all, and the various modifications and changes in technic that have been described by a number of writers in the past decade owe their origin to the universal feeling of discontent with results in direct hernia. Where many modifications and new operations are advocated, it can be assumed that no one method is entirely satisfactory. And today the problem of hernia resolves itself largely into the attempt at reducing the recurrence rate of direct hernia. Rectus transposition, imbrication of the external oblique aponeurosis, and other modifications of the ordinary Bassini all have their advocates. There is, however, in the writer's opinion, no one technic applicable to all direct herniæ. The method of approaching each case as an individual, and reserving the decision as to the type of closure to be performed until a survey of the patient's available tissues has been made, seems to be far preferable to the use of any one variation. In the 280 cases here considered, rectus transplant and imbrication of the oblique have been the two modifications most often utilized. An attempt to evaluate their relative merits will be made in a later communication. Suffice it to say here that, many direct herniæ will resist the most skillful and painstaking operative efforts. The occupation of the patient, his age, the size of the hernia, the condition of his abdominal musculature, and the degree of symptoms caused by the hernia, should all be considered before plunging the patient into an operation which, if it fails, may leave him with a recurrence larger than his original hernia and less amenable to treatment with a truss. Downes [2] has well stated that many a direct hernia is better left unoperated. However, it is the writer's conviction—based on a small number of cases in this group—that the "living suture" of Gallie,[3] either in association with a typical Bassini or with some modification (Halsted-Andrews, Schley,[4] Stetten,[5] etc.), offers the patient the greatest possible likelihood of a permanent cure; and that when the technic has been mastered and generally adopted, this method will become the operation of choice in selected cases of direct inguinal hernia. Gallie's method forms a bulwark of strips of fascia taken from the thigh and so woven across the defect as to close it without attempting to pull its edges together. It can best be compared to the darning of a hole in a stocking. We have tried this method in a few cases but a sufficient length of

time has not yet elapsed to draw conclusions as to end-results. It seems, however, a most rational procedure. It should be reserved for carefully selected cases as it adds appreciably to the duration of the operation. For this and other reasons, it has not seemed to us advisable in obese subjects or those past middle age. Primary union of the incision on the thigh from which the fascia is obtained has taken place in all the cases we have done by this method. Gallie's own results show 60 cases operated upon more than two years ago with no recurrences. After a sufficient number of cases have been operated upon by this method, and a sufficient period of time has elapsed, the results of the " living suture " method in our cases will be the subject of a later report.

3. *In Female Child.*—No case has ever been operated upon at this institution.

4. *In Female Adult.*—Three cases were operated upon, two were traced, with one recurrence, or 50 per cent.

Though rare, when present this variety of hernia offers as apparently great an obstacle to permanent cure in females as in males, the percentage-recurrence in the entire 10,000 series being 12.5 per cent. of 24 cases.

Direct and Indirect Hernia (Saddle-bag Type)

1. Male child ..... 2 cases, neither traced.
2. Male adult ..... 98 cases with 5 recurrences, or 11.9 per cent. of 42 followed cases.
3. Female child ..... 0 cases observed.
4. Female adult ..... 1 case, not followed.

This condition is essentially one of adult life and is rarely observed in females. Its recognition is quite important as in those cases in which the indirect sac is more pronounced, failure to find the direct portion will almost inevitably lead to a recurrence. For the treatment of this variety of direct hernia—for so it should be regarded—we adopt the principles applicable to simple direct hernia-repair. The disposition of the sac, however, is a point worthy of consideration. The most convenient and logical procedure is that suggested by Downes, namely, conversion of the two sacs into one, by peeling the deep epigastric vessels off the isthmus with the finger introduced through the opened indirect sac into the direct. Thus one large and broader " indirect " sac is produced and the high ligation effected as in any indirect hernia. Should it be difficult to perform this manœuvre, as when operating under local anæsthesia, one may divide the vessels between ligatures.

Femoral Hernia.—1. *In Male Child.*—Eleven cases with no recurrence but 10 of the 11 cases were not traced.

2. *In Male Adult.*—There were 32 operations (11 followed) with one recurrence, or 9.09 per cent.

3. *In Female Child.*—Ten operations with no recurrence, only two cases having been followed.

4. *In Female Child.*—There were 48 operations with 2 recurrences, or 14.2 per cent., based on the 14 followed cases.

TABLE I

Summary of 3000 Consecutive Herniotomies.

Type	No. of operations	6 mos.—1 yr. followed	1 yr. or over followed	Total	Recur.	% Recur.
Inguinal male child indirect	1013	89	174	263	0	0
Inguinal male adult indirect	1155	103	229	332	28	8.7
Inguinal male child direct	4	0	1	1	0	0
Inguinal male adult direct	280	28	57	85	14	16.4
Inguinal male child direct and indirect	2	0	0	0	0	0
Inguinal male adult direct and indirect	98	19	23	42	5	11.9
Inguinal female child indirect	124	10	12	22	0	0
Inguinal female adult indirect	113	7	16	23	1	4.4
Inguinal female child direct	0	0	0	0	0	0
Inguinal female adult direct	3	2	0	2	1	50
Inguinal fem. child direct and indirect	0	0	0	0	0	0
Inguinal fem. adult direct and indirect	1	0	0	0	0	0
Femoral male child	11	1	0	1	0	0
Femoral male adult	32	8	3	11	1	9.09
Femoral female child	10	0	2	2	0	0
Femoral female adult	48	4	10	14	2	14.2
Umbilical male child	11	0	3	3	0	0
Umbilical male adult	11	2	1	3	0	0
Umbilical female child	9	2	0	2	0	0
Umbilical female adult	23	6	4	10	1	10
Ventral male child	5	2	2	4	0	0
Ventral male adult	14	2	3	5	1	20
Ventral female child	1	0	0	0	0	0
Ventral female adult	18	1	3	4	1	25
Epigastric male child	2	1	1	2	0	0
Epigastric male adult	9	2	2	4	1	25
Epigastric female child	0	0	0	0	0	0
Epigastric female adult	3	0	2	2	1	50
	3000	289	548	837	57	6.8

* The floating character of the foreign-born patients makes circular follow up letters a rather ineffectual method of recalling them for periodic examinations and is an expense to the hospital. Personal contact between operator and patient is more productive of good results. The earlier series reported by Bull and Coley showed a much higher proportion of cases followed. We feel that the percentage in this series traced longer than six months, (837 in 3000 or about 27 per cent.,) is far too low and efforts are now being made to materially improve this.

In view of the numerous communications advocating the inguinal approach for femoral herniotomy, it might be well to state our position. Though we have occasionally tried out this method, we have used and continue to use the femoral approach with closure by purse-string of the femoral canal, first, because it has seemed a far simpler procedure and has served us well, there being but 3 recurrences in 28 followed cases; and second, because none of the advocates of the inguinal route has thus far been able in a conclusive series to show a lower percentage of recurrence. Should statistics ultimately show the inguinal route to be productive of a higher percentage of cures, the more complicated method would then become justifiable, as a routine procedure. In strangulated femoral hernia, the inguinal approach is admirable.

Umbilical Hernia.—1. *In Male Child.*—Eleven cases with no recurrence, 3 cases having been followed.

2. *In Male Adult.*—Eleven cases with no recurrence, 3 cases having been followed.

3. *In Female Child.*—Nine cases with no recurrence, only 2 cases having been followed.

4. *In Female Adult.*—Twenty-three cases with one recurrence, or ten per cent. of ten followed cases.

The usual type of Mayo overlapping operation has proved satisfactory in these cases. We have found that the majority of umbilical herniæ in very young children respond to strapping with the button, and hence, operation is seldom performed under the age of twelve years. If strapping fails, we see no object in postponing operation thereafter.

Ventral Hernia.—1. *In Male Child.*—Five cases; 4 followed with no recurrence.

2. *In Male Adult.*—Fourteen cases; 5 followed, with 1 recurrence or 20 per cent.

3. *In Female Child.*—One case, not followed.

4. *In Female Adult.*—Eighteen cases with 1 recurrence or 25 per cent. of 4 followed cases.

The usual lateral overlapping operation with suture of the abdominal wall in layers, where possible, has been the customary procedure.

Epigastric Hernia.—1. *In Male Child.*—Two cases, both followed, no recurrence.

2. *In Male Adult.*—Nine cases, 4 followed, with 1 recurrence or 25 per cent.

3. *In Female Child.*—No cases.

4. *In Female Adult.*—Three cases, 2 followed, with 1 recurrent or 50 per cent.

Double Hernia.—In compiling the statistics for this paper, it seemed of interest to determine the number of instances in which a hernia was present on both sides; these findings are given in Table II. Attention is called to the fact that, while these figures represent the cases operated upon in which a

hernia was found on both sides at the time of operation, there are also a number of cases, particularly in children, in which, after operation on one side, a hernia has developed within a year on the opposite side, which was not detected at the time of the original operation. Some surgeons believe the development of an indirect inguinal hernia on the opposite side in children is of so frequent occurrence that, in finding this type of hernia on one side, they immediately operate upon the opposite apparently normal side often finding a small incipient hernia which had escaped detection during the routine examination prior to operation. It is not our opinion that a double operation

TABLE II

Incidence of Double Hernia in 3000 Cases.

Double indirect male adult	255
Double indirect male child	210
Double direct male adult	82
Double direct male child	0
Double direct and indirect male adult	22
Double direct and indirect male child	0
Double indirect female adult	24
Double indirect female child	27
Double direct female adult	0
Double direct female child	0
Double direct and indirect female adult	0
Double direct and indirect female child	0
Double femoral male adult	5
Double femoral male child	2
Double femoral female adult	9
Double femoral female child	0
Total	636
Incidence	26 per cent.

should be routinely performed in all unilateral indirect inguinal herniæ in children; and we believe that if a large series of cases were considered, the percentage in whom a second operation was made necessary by the subsequent development of a hernia on the opposite side, would be very much smaller than the percentage in whom a second hernia had not developed.

Undescended Testis.—As the subject of undescended testis will be fully discussed in a later communication, we will merely mention here that, in the 3000 cases of hernia, there were 39 operations for undescended testis in adults, and 90 in children. The undescended testis was right-sided in 32 instances in adults, and 41 in children; left-sided in 7 of the adults, and 49 of the children. The constant finding of an associated congenital hernia, either fully developed or potential, needs only to be mentioned in passing.

Recurrences.—At present, the chief interest in the subject of hernia lies, not in a consideration of successful operations, but rather in an analysis of failures. It would seem that there are two distinct sets of factors which may be held accountable for recurrence following an operation for hernia. The first, comprises factors inherent in the individual subject, *e.g.*, the age, occupation, size of the hernial orifice, lack of development of the abdominal muscles, especially deficient conjoined tendon, presence of adipose tissue in abnormal

amounts or abnormally placed, presence of a sliding hernia or a strangulated hernia with the patient in a critical condition. These factors are for the most part beyond the control of the operator and in so far as they play an important part in the causation of recurrences, just so far will operative efforts continue to meet with ultimate failure. It is particularly in individuals who present a combination of several of these factors that recurrences are most certain to follow. Some should, undoubtedly, be advised against operation, and more care in selecting cases for operation will reduce the rate of recurrence in this group and work for the ultimate benefit of the patient. In the second group may be placed those factors which do not properly belong to the patient himself and over which the surgeon has or should have control. Here we include failure to recognize and satisfactorily obliterate the sac or both sacs where the hernia is of the saddle-bag type. This is the probable explanation of several recurrences which were noted directly the patient was allowed out of bed. The presence of infection markedly predisposes to a recurrence and, indeed, may be looked for, should it involve the deep layer of sutures. As a corollary the use of non-absorbable suture material which predisposes to sinus formation is a factor in recurrence. Such apparently trivial matters as the length of time the patient is kept in bed, the failure to provide support by careful adhesive strapping or spica bandage during the healing period, or to forestall the development of post-operative bronchitis or pneumonia with its attendant cough—these may turn the scale and favor the formation of a recurrence despite faultless operative technic.

Here, too, may be raised the question of re-operation on a hernia that has once recurred; particularly in inguinal hernia there is much to persuade one that many patients with recurrence had better wear a truss than undergo a second operation. This, of course, refers to the direct recurrences more especially, though, as a matter of fact, the majority recur as direct and not as indirect. It is interesting to note that of 47 recurrences of inguinal herniæ (direct or indirect) in male adults in this last series, 6 or 12.7 per cent. had followed previous recurrences. In carefully selected cases, recurrent herniæ treated by some form of fascial transplant, the most rational of which seems to be the "living suture" repair of Gallie, will probably be successful.

Interval between operation and recurrence.—In the 57 recurrences in this series of consecutive herniotomies, the average elapsed time between operation and recurrence was 7.1 months. The longest elapsed time was 31 months. There were five instances in which recurrence was noted during the first month after operation: In one of these cases, a direct sac was presumably overlooked in the repair of an indirect inguinal hernia; in another, an epigastric hernia, the sutures gave way while the patient was still in the hospital; a third was recurrent when operated upon and a second recurrence promptly supervened. Although we attempt to follow all cases for five years or more, and some of the earlier ones have been traced over twenty years, it is evident that if cases are observed for two years, the majority of recurrences will be detected.

TABLE III.

Classification of Fifty-seven Recurrences in 3000 Cases

No.	Sex	Age	Type of hernia	Interval	Type of op. performed	Type of recurrence
1	M.	47	L. D. I. H. recur.; R. O. I. H.	3 mos.	Bassini	L. recurrence, prob. as direct.
2–3	M.	56	D. D. I. H.	8 mos.	Bass., imbrica. Trans. f.	Double recurrence.
4	M.	24	R. I. Superf., Und. T.	18 mos.	Bass., cord not transpl.	R. recurrence prob. indirect.
5	M.	37	D. O. I. H., double varicocœle	18 mos.	Bass. with double varicocœle op.	R. recurrence.
6	F.	35	R. O. I. H.	5 mos.	Bass. round lig. not tr.	R. recurrence.
7	M.	55	R. D. and O. I. H.	4 mos.	Bassini	R. recurrence prob. as indirect.
8	M.	37	L. O. I. H.	1 yr.	Bassini	L. recurrence prob. as direct.
9	M.	40	R. O. I. H. and L. D. I. H.	16 mos.	R. Bass.; L. Bass., R. T.	R. recurrence.
10	M.	17	D. O. I. H.	5 mos.	Bassini	R. recurrence.
11	M.	53	L. O. I. H.	2 mos.	Bassini (good closure)	L. recurrence.
12	M.	42	D. D. I. H.	Immed.	Bass., imbrica. Ext. Obl.	L. recurrence.
13	M.	30	L. O. I. H.	31 mos.	Bassini	L. recurrence at int. ring.
14	M.	30	D. D. I. H.	2 mos.	Bassini R. T., imbric. Ex. Obl.	R. recurrence as a direct.
15	M.	42	D. D. I. H.	5 mos.	Bassini with R. T.	L. recurrence.
16	F.	65	Umbil. H.	6 mos.	Mayo overlapping	Large recurrence.
17	M.	40	D. D. and O. I. H.	6 mos.	Bassini with R. T.	R. recurrence.
18	M.	42	L. O. I. H.	2 mos.	Bassini	L. recurrence faint impulse.
19	M.	30	R. D. I. H. and L. O. I. H.	6 mos.	Bassini	R. recurrence as direct
20	M.	45	R. O. I. H.	9 mos.	Bassini	R. recurrence.
21	M.	44	R. O. I. H.	4 mos.	Bassini	R. recurrence as direct.
22	M.	40	Ventral H.	7 mos.	Mayo overlapping	Recurrence throughout incision.
23	M.	48	L. O. I. H.	2 mos.	Bass., imbric. Ex. Obl. over cord	Recurrence at lower angle.

TABLE III.—*Continued.*

Classification of Fifty-seven Recurrences in 3000 Cases.

No.	Sex	Age	Type of hernia	Interval	Type of op. performed	Type of recurrence
24	M.	27	L. O. I. H.	11 mos.	Bassini	L. recurrence.
25	M.	40	D. D. I. H.	1 yr.	Bassini, rectus imbr. Ex. O.	R. recurrence.
26	M.	48	L. D. I. H.	1 mo.	Bassini	L. recurrence.
27	M.	42	L. D. I. H.	2 mos.	Bassini and Halstead	L. recurrence.
28	M.	29	L. O. I. H.	8 mos.	Bassini	L. recurrence.
29	M.	45	R. O. I. H. recur.	3 wks.	Bass. R. T. and imbr. Ext. O.	R. recurrence lower angle, D?
30	M.	35	L. D. and O. I. H., R. O. I. H.	Immed.	Bassini	R. recurrence on getting out bed.
31	M.	36	D. D. and O. I. H.	18 mos.	Bassini with R. T.	L. recurrence.
32	M.	57	D. D. I. H. recur.	2 mos.	Bassini with R. T.	L. recurrence, slight.
33	M.	43	V. H., R. D. I. H.	21 mos.	Bassini with R. T.	R. recurrence, D. slight.
34	M.	21	L. O. I. H., Und. Testis	17 mos.	Bassini, cord not tr.	L. recurrence.
35	M.	32	R. O. I. H.	3 mos.	Bassini	R. recurrence.
36	M.	32	D. D. and O. I. H.	9 mos.	Bassini?	L. recurrence.
37	F.	53	Ventral H.	6 mos.	Vertical overlapping	Ventral.
38	F.	55	R. D. I. H., L. O. I. H.	6 wks.	Bassini R. L. N. T.	R. recurrence.
39	M.	40	D. O. I. H., R. Und. Testis	6 mos.	Bassini	L. recurrence.
40	M.	48	D. D. L. H. recur.	3 mos.	Bassini, imbr. of Ext. O.	L. recurrence.
41	F.	52	Epigastric H.	4 mos.	Mayo overlapping	Recurrence throughout inc.
42	M.	29	L. D. I. H.	11 mos.	Bass. with lower two sut. incl. rectus; Imb. Ex. O.	L. recurrence.
43	M.	38	D. O. I. H.	13 mos.	Bassini	R. recurrence.
44	M.	51	D. O. I. H.	4 mos.	Bassini	R. recurrence.
45	M.	47	R. recur. I. H., indirect	10 mos.	Bassini, rectus transp.	R. recurrence middle of incision.
46	F.	38	L. F. H.	7 mos.	Two purse-strings	Recurrence.
47	M.	30	D. O. I. H.	4 mos.	Bassini C. N. T.	L. recurrence.

TABLE III.—*Continued.*

Classification of Fifty-seven Recurrences in 3000 Cases.

No.	Sex	Age	Type of hernia	Interval	Type of op. performed	Type of recurrence
48	F.	48	D. F. H.	7 mos.	Two purse-strings	R. recurrence.
49	M.	38	D. O. I. H.	4 mos.	Bassini	R. recurrence.
50	M.	45	R. O. I. H.	4 mos.	Bassini	R. recurrence, direct, small.
51	M.	38	L. O. I. H., recur.	10 mos.	Bass. with rectus trans.	L. recurrence.
52	M.	55	D. F. H.	1 mo.	Two purse-strings Imbr. Ext. Obl.	R. recurrence.
53	M.	63	R. O. I. H. recur.	6 wks.	Bass. apon.; vas and vessels emerging separately	R. recurrence slight at lower angle.
54	M.	39	E. H.	Immed.	Vert. overlap. Mayo	Recurrence.
55	M.	24	R. O. I. H.	1 mo.	Bassini	R. recurrence, direct.
56	M.	42	D. D. and O. I. H.	2 yrs.	Bass. with Imb. Ext. Obl.	L. recurrence, direct?
57	M.	16	D. O. I. H. and Ventral H.	2 yrs.	Bass, lateral overlap.	R. recurrence.

Age at Operation.—The average age at operation in the recurrent cases was 38.1 years. The females averaged 45.1 and the males 38.6. The youngest recurrent case was 16 years, and the oldest, 65 years; the former, a double indirect inguinal and ventral hernia in a male, and the latter, an umbilical in a female.

Classification of Recurrences

Indirect inguinal, 8 in 1268 adult operations
Direct inguinal, 16 in 283 adult operations
Direct and indirect, 5 in 99 adult operations
Femoral, 3 in 80 adult operations
Ventral, 2 in 32 adult operations
Epigastric, 2 in 12 adult operations
Umbilical, 1 in 34 adult operations

No recurrences in children.

Explanation of Recurrences.—Since a recurrence is often the result of a combination of several factors, any discussion may be considered theoretical; moreover, we are hampered by the fact that in most cases the records fail to state the opinion of the operator as to the cause of failure. This is inevitable in a follow-up system where the cases are examined by members of the entire staff at random, though this method probably results in more unbiased examinations. In six instances, recurrence followed operations for previous recurrences; in two, the hernia was of the sliding variety, with the implied failure to obtain a high closure of the sac. Infection and drainage of the wound was the probable cause in two cases, and in two others, associated with undescended

testis, the cord was not transplanted. In one instance, a direct sac was probably overlooked, and two were attributed by the operator, to poor closure due to deficient musculature.

Disposition of Recurrences.—In general, the decision as to treatment of recurrent cases depends upon the type and size of the recurrence, the age and occupation of the patient, and the condition of the musculature. For the most part, small direct recurrences are generally given a well-fitting truss. A few are given the choice of operation, though it is not urged upon them.

TABLE IV

Deaths in 3000 Consecutive Herniæ

No.	Sex	Age	Type of hernia	Operation performed	Time of occurrence of death	Cause of death
1	F.	50	Umbilical; strang.	Mayo overlapping	Died on table	Anæsth., shock, obstruction.
2	M.	67	R. O. and Direct Ing.	Bassini, rectus trans.	5th post-op. day	Post-anæs., pneumonia.
3	M.		L. D. I., L. Hydrocele	Bassini, rectus trans.	5th post-op. day	Pulmonary embolus.
4	F.	55	Post-op. Ventral	Lateral overl. modified	4th post-op. day	Paralytic ileus.
5	M.	59	R. O. Scrotal, recur.	Bass., Imbrica. oblique	14th post-op. day	Renal insuf. Hypostа., Pn.
6	M.	3	L. O. I. H. scrotal	Bassini	2nd post-op. day	Acidosis.
7	M.	25	L. O. I. H. recur.	Bassini, rectus transp.	Not stated	Post-op. pneumonia.
8	M.	43	R. O. I. H.	Bassini	4th post-op. day	Post-op. pneumonia.
9	M.	81	Double D. I. H.	Bassini under local an.	2nd post-op. day	Pulmonary embolism.
10	F.	32	R. Femoral H.	Mod. Cushing; 2 purse-s.	5th post-op. day	Pulmonary embolus.
11	M.	6	Double O. I. H.	Bassini	3rd post-op. day	Acidosis; convuls.

Two cases were re-operated upon elsewhere, one having become strangulated. Though our records are incomplete in this respect, it would seem that about twenty-five per cent. of the recurrences were re-operated upon by us, the remainder being given some form of retentive apparatus. Some have undoubtedly sought re-operation elsewhere. As we become more familiar with the Gallie " living suture " technic, it is not unlikely that the field of operability of these recurrent cases will be greatly extended; and already we are offering operation to patients whom we would have felt had but slight likelihood of a cure by any other procedure.

Type of Operation Performed in Cases which Recurred

Inguinal Hernia.—Simple Bassini with cord transplantation	25 times
Simple Bassini with cord not transplanted	2 times
(Both cases associated with und. test.)	
Simple Bassini with round ligament not transplanted	2 times
Bassini with rectus transplantation	9 times
Bassini with imbrication of ext. obl. aponeurosis	7 times
Bassini with rectus transposition and imbrication of ext. obl. aponeurosis	3 times
Femoral Hernia.—Modified Cushing purse-string	4 times
Ventral Hernia.—Vertical overlapping	1 time
Mayo overlapping	1 time
Epigastric Hernia.—Mayo overlapping	2 times
Umbilical Hernia.—Mayo overlapping	1 time

Technical Considerations.—The methods of operating upon hernia used at the Hospital for Ruptured and Crippled have been described so often that a detailed consideration of the steps in technic is unnecessary here. In brief, since 1890, the great majority of cases of inguinal hernia have been operated upon by the Bassini method, with slight modifications described by Coley (William B.) in Keen's Surgery, 1908. With the exception of two or three cases operated upon in 1890, in which silk sutures were used, chromicized kangaroo tendon has been used for the buried sutures during the entire period. In direct hernia, a number of different methods or variations of technic have been employed at different times. At present we are inclined to favor the transplantation of the cord to a position superficial to the external oblique, as in the Halsted operation, with the overlapping of the external oblique aponeurosis as advocated by Champonniere and Andrews, and recently modified by Schley and Stetten. If this method be adopted, and in addition, fascial sutures (Gallie) be utilized, we believe that the results in these difficult cases will be very markedly improved.

Local Anæsthesia.—There is no doubt of the desirability of using local anæsthesia for strangulated hernia, where the patient's condition is apt to be such that the general anæsthetic may turn the balance against recovery. And it has widened the field of operability to include otherwise bad risks; the diabetic, the cardiac, the elderly patient. However, in children, and in most adults where a definite contra-indication does not exist, we prefer a general anæsthetic, generally gas-oxygen with a minimal amount of ether in adults, and drop ether by open cone method in children. The results are most satisfactory from the point of view of high ligation of the sac, tightness of closure, and duration of the operation. In short, though novocaine anæsthesia has a distinct and useful place in herniotomy, it has not become a routine procedure with us. Morrow states that he has used it for fifteen years as a routine measure, believing that in addition to its other advantages, it eliminates the recurrences that may be due to vomiting and straining during recovery from a general

anæsthetic. It has been our experience that, where gas and oxygen with a minimal amount of ether is used, post-operative emesis is not pronounced.

Mortality.—There were eleven deaths or a percentage mortality of 0.37. In ten cases, death occurred between the first and fifth post-operative days. The causes of death represent the usual surgical hazards. In only one instance was advanced age of significance; this patient being a male of eighty-one years, whose Bassini repair for double direct inguinal hernia had been performed under local anæsthesia, and who died of pulmonary embolus on the second post-operative day. The one case operated upon for strangulation was a huge umbilical hernia, and death which occurred on the table, was due to a combination of shock, toxæmia from ileus, and the general anæsthetic. The anæsthetic is the only factor which might better, perhaps, have been eliminated, substituting instead, local. As a general rule, strangulated hernia in adults should be dealt with under local anæsthesia.

SUMMARY

1. Three thousand consecutive recent herniotomies have been classified according to age, sex, and type of hernia. Recurrence rates in each group have been compiled, based on 837 cases actually followed up.

2. Causes of recurrence may be arbitrarily divided into: (a) Those factors which are inherent in the individual patients, *e.g.*, age, occupation, size and type of hernia, development of abdominal musculature, presence of adipose tissue, etc. The complete or partial absence of conjoined tendon is the main surgical problem that must be met. (b) Factors over which the surgeon has control, *e.g.*, failure to recognize and dispose of hernial sac, wound infection, use of non-absorbable suture material, closure under tension, failure to support operative wound by proper dressings, and to forestall post-operative bronchitis and pneumonia with their attendant cough by appropriate preventative measures, allowing the patient out of bed too soon, etc. The exercise of more careful judgment in selecting cases for operation, basing the decision on a consideration of the patient's possible factors of failure on the one hand, and more attention to the details of operative technic and post-operative care on the other, will tend to reduce recurrence rates.

3. Recurrence in children should be a rare sequel to a properly performed operation. In 1191 operations, for all varieties of hernia, 300 of which were followed, we have been unable to find a single recurrence.

4. Follow-up examinations of all cases is becoming the rule in most metropolitan clinics. It has been the rule in the Hospital for Ruptured and Crippled for thirty years. Recurrence rates not based upon a follow-up examination should be discounted.

5. Direct hernia is rare in the female and in children under fifteen years; three cases of the former, and four of the latter (all males) were found in this series. The cure of direct hernia represents a distinct problem. Many

are best managed by some modification of the Bassini operation. A certain proportion present musculo-aponeurotic deficiencies which render the likelihood of a permanent cure slight, and in this group, operation should be withheld. In the exercise of more careful judgment in selecting cases for operation, as Morrow [6] has stated, lies the hope for better end-results, rather than in the adoption of new procedures based upon new principles. "Saddle-bag" or direct and indirect hernia should be considered as a variety of direct hernia and so treated. Removal of both sacs is essential. Our series of 280 direct hernia in the male adult, of which, 85 cases have been followed, shows 14 recurrences or 16.4 per cent. The "living suture" repair of Gallie has a wide usefulness in this variety of hernia and may become the method of choice.

6. There is lacking, thus far, any conclusive evidence that femoral hernia repair by the inguinal route is productive of better results than have been obtained by the simpler method.

7. Cases that have once recurred will be more apt to recur again, and the second recurrence may be larger and less amenable to truss treatment; therefore, the question of re-operation should be most carefully considered, and in every case, it should not be advised. Thirteen per cent. of the fifty-seven recurrences in this series, had followed operation for previous recurrence. Gallie's operation, using "living sutures" has a distinct place in this group.

8. Consideration of the available tissues for repair should determine the type of operation to be used in each individual case. The use of a standardized technic for all cases is to be deprecated.

9. Local anæsthesia has extended the field of operability to include the aged and those suffering from intercurrent disease which, in itself, contraindicates the use of a general anæsthetic. It is strongly urged in cases of strangulated hernia in adults. However, as a routine, we favor for adults, gas-oxygen with the addition of a small amount of ether if necessary; and for children, ether by drop method and open cone.

10. We subscribe to Morrow's statement that many surgeons consider the operative cure of hernia a simple procedure, and agree with him that it requires sound surgical judgment and considerable technical skill.

The writer acknowledges the courtesy of Dr. John B. Walker and Dr. William B. Coley in permitting him the privilege of compiling these statistics based on the records of the First and Second Hernia Divisions of the Hospital for Ruptured and Crippled.

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THE SURGICAL ASPECTS OF XANTHOMA TUMORS

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Since Rayer [1] in 1836 first described the yellow plaques of the eyelids, and Lebert [2] in 1845 mentioned a peculiar yellowish fatty substance found in certain tumors of the soft parts, xanthomatous lesions have given rise to many interesting studies by dermatologists, chemists, pathologists and in more recent years by surgeons. At the present time two well-defined clinical forms are recognized: (1) The so-called *xanthelasma*, a local lesion confined to the eyelids and (2) *xanthoma multiplex*, which is a generalized form manifesting itself by widespread cutaneous lesions. In addition there are solitary forms occurring as xanthomatous tumors, about which, as we shall see, there is considerable dispute as to classification.

Fig. 1.—Xanthoma of foot. (Cross-section.) This was a well encapsulated tumor arising from the flexor tendon sheaths, presenting a brilliant yellow-orange color on section.

Xanthelasma, the form most commonly met with, is characterized clinically by the presence of multiple, small, soft, yellowish, chamois-like patches on the eyelids. They are often symmetrically arranged and appear as a rule near the inner canthus of the eye. They occur almost invariably in persons beyond middle life, develop slowly, are not associated with disease elsewhere in the body, and remain unchanged for many years. For the histology of these lesions we are especially indebted to Pollitzer and Wile [3] and Waldeyer [4] who have shown that they represent a degenerative process within the muscle tissue of the eyelid and appear histologically as an accumulation of large polyhedral foamy cells which have undergone a peculiar fatty degeneration.

Xanthoma multiplex, a form more rarely seen, but even more striking than the first, is a generalized manifestation of an eruptive disease, appearing cutaneously as yellowish lesions varying from papules, nodules and tubercles to flat infiltrated plaques or fine yellowish lines. They occur in small groups often symmetrically arranged or in large numbers, especially about the

joints—the shoulders, elbows, hips and knees, but not uncommonly widely distributed over the trunk and extremities. It is met with particularly in young people and may even be congenital. A close study of these cutaneous lesions by many investigators since Virchow [5] first described them as degenerated forms of a connective tissue neoplasm, has brought to light unmistakable evidence that they represent a connective tissue reaction toward a retained foreign material, and that they bear a definite relation to diseases of metabolism. Examination of early lesions by Pinkus and Pick,[6] Pollitzer and Wile,[7] Macleod [8] and others has shown that the initial phase consists in a dilatation of the cutaneous capillaries, a proliferation of the perivascular endothelium and a deposit within the tissue spaces of a finely divided lipoid substance which in later stages is absorbed by the endothelial and connective tissue cells as a foreign material. This gives rise to the formation of large polyhedral cells with small nuclei and granular cytoplasm and is accompanied by a reaction on the part of the surrounding tissues resulting in the formation of large multinuclear foreign body giant cells and an active proliferation of the fibrous tissue. The frequent

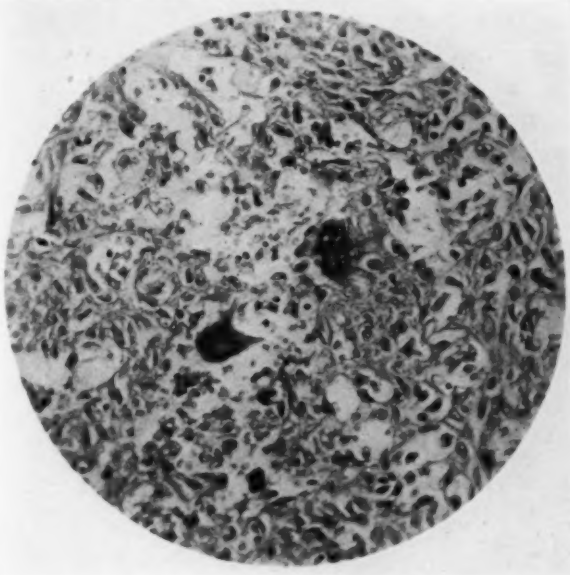

FIG. 2.—Xanthoma of the foot. Low power showing proliferation of fibrous connective tissue and large multinuclear foreign body giant cells. (Magnification 240 diameters.)

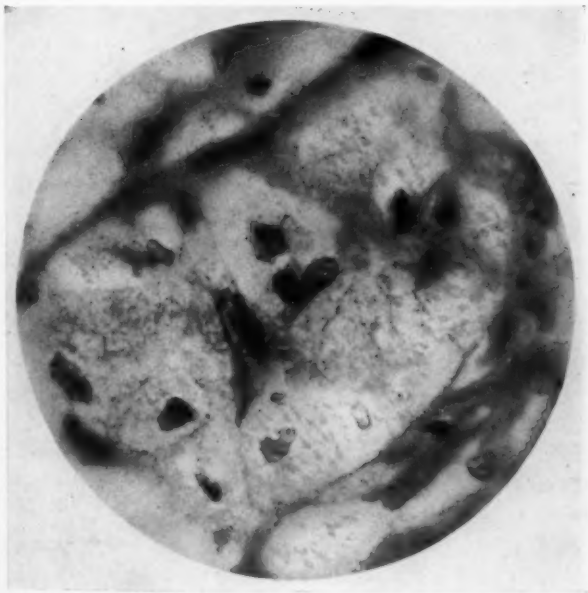

FIG. 3.—Xanthoma of foot. High power view showing typical "foam" cells. (Magnification 1200 diameters.)

proximity of these lesions to the joints is explained on the basis of increased intracapillary tension resulting from constant mobility of the tissues. The exact nature of this lipoid substance giving rise to these lesions has been the object of many studies, and has been definitely identified by Pinkus and Pick,[9] Pringsheim,[10] Pollitzer and Wile,[11] Darier,[12] Burns,[13] Smith[14] and many others. It consists of a cholesterol fatty acid ester which is doubly refractive to polarized light. In fresh tissues fixed in formalin the lipoid granules appear as small yellow-red globules when stained with sudan III, and assume a gray color with osmic acid. When fixed in alcohol they are readily dissolved, and leave behind small vacuoles which appear microscopically like foam, and have given rise in the literature to the term "foam cells" or "xanthoma cells." The detection of this lipoid and the close association of xanthoma multiplex with jaundice and diabetes have been the stimulus for a vast amount of research devoted to the solution of the fundamental chemical problems concerned. While a critical review of this work is not essential to the subject under discussion, yet a brief summary of the main features seems necessary before we may have a clear perception of the meaning of xanthomatous tissues in the forms in which surgeons most commonly see them.

FIG. 4.—Picture made six months after removal of xanthoma tumor (Case I). There is no evidence of recurrence.

Cholesterin, an integral part of all body tissues, exists normally in the blood serum in a concentration of about .16 per cent. It is derived partly from the food, but mainly from the internal secretions of the various tissues. It is eliminated through the bile either in the normal state or in the form of cholalic acid. It is increased physiologically during menstration, pregnancy, and in the puerperal state, and to its presence has been attributed the severe vomiting during the early months of the child-bearing period. Its characteristic large flat crystals may be found in any tissue in which cells are undergoing slow destruction and where absorption is poor, as in atheromatous patches of the

blood-vessels, encapsulated caseous areas, old infarcts or hæmatomas. Its ratio in the blood is markedly increased in Bright's disease and particularly in icteric conditions due to obstruction to its normal channel of elimination—the common bile duct.

According to Wells [15] *fat* in body tissues is derived (1) from food and (2) from carbohydrates by a synthetic process. If from proteins at all it is probably due to the presence within them of carbohydrate groups. It is utilized and transported in the form of its two primary constituents, fatty acids and glycerol. It enters and leaves the cell in this condition, being split or combined by the action of the tissue lipase. Normally there is little visible fat in the cells of the parenchymatous organs because they are constantly being used up by the oxidation of the glycerol and fatty acids by the intracellular oxidases. Where there is abundant lipase and little oxidative activity, as in the areolar tissues and liver, fat accumulates in large amounts. In a true fatty infiltration, which is dependent upon decreased oxidation, the fat accumulates within the cells as a single large droplet; whereas in a true fatty degeneration, which is brought about by poisons, the fat droplets lie well distributed in the granular debris. The accumulation of fat in dead tissue such as an anemic infarct, is due to the action of the cell lipase upon the fatty acids and glycerol which are diffused in from the periphery into an area deprived of oxygen. The amount of fat actually present in a tissue cannot be accurately judged by the histological appearance of the fat within the cells, because a certain portion of the fat is held in such firm combination that it cannot be demonstrated by staining methods. This fixed material is not simple fat, but lecithin and cholesterol compounds. In 1902, Kaiserling and Orgler [16] described under the name "Myelin" certain intracellular droplets that may be found in the adrenal cortex, in amyloid kidneys, penumonic exudates, tumor cells, retrogressive thymus tissue, corpus luteum, and atheromatous patches of arteries. This material differs from ordinary fat in being doubly refractive (anisotropic) when viewed with the Nicoll prisms and in staining but slightly gray with osmic acid. These myelins, according to Wells, are probably mixtures of lipoids in which cholesterol esters are very prominent. This work

FIG. 5.—Case II. Cystosarcoma-phylloides of the breast.

has been corroborated by Dunin-Karwicka [17] who has shown that this same material is found in the cortical cells of the adrenal gland, lutein cells of the ovary, and is seen pathologically in the atheromatous arteries, actinomycosis, chronic inflammatory areas, and is the chief constituent of hypernephromas and xanthomatous tumors.

In view then of the conditions under which cholesterol esters are found in the body it is only reasonable to suppose that xanthomatous tissue may not only be widely distributed, but would be most abundantly found in diseases associated with a lipemia or a hypercholesterinemia, and this supposition is readily confirmed by clinical observation. Diabetes mellitus, as is well known, is almost invariably accompanied by a lipemia and with it we often see generalized xanthomatous lesions. Mook and Weiss [18] have recently reported three cases of generalized xanthoma associated with diabetes and two with a hypercholesterinemia. Rosenbloom [19] in 1913 and Burns [20] in 1920 made determinations by the Bloor method, the latter finding three times the normal cholesterol content of the blood in xanthoma multiplex. Similar findings have been reported by Yamakawa and Kashuwabara.[21] Pinkus and Pick [22] have studied symmetrical xanthoma lesions in a patient intensely jaundiced and Kaposi [23] has stated that 56 per cent. of the generalized forms of xanthoma are associated with icterus. Perhaps the most interesting and instructive report, however, has recently come from Weidman and Freeman [24] who observed widespread xanthomatous lesions of the internal organs and an extreme cutaneous involvement in a boy who had died after a long-continued jaundice. The seat of the original trouble was a biliary cirrhosis, which in turn had led to a hypercholesterinemia and consequent xanthomatous changes in the tissues. The hilum of the liver, the interstitial tissue of the lung alveoli

FIG. 6.—Cystosarcoma-phylloides, (Case II). Cross-section of the tumor showing the yellow area A of xanthoma tissue. This tumor was in all respects benign.

the bones of the cranium, the meninges and stalk of the hypophysis all showed histologically the typical foam cells, foreign body giant cells and grossly had the brilliant yellow color which are characteristic of xanthoma lesions.

In view of the fact, that, except when associated with a hypercholesterinemia or a lipemia, the presence of xanthomatous tissue is indicative of a degenerative process peculiar to endothelial or connective tissue cells, it is quite evident that a consideration of xanthomattous tumors from the surgical point of view, must necessarily be confined to those in which one or both of these types of cell predominate. In the literature there has been much difference of opinion regarding these tumors and not a little confusion as to their classification, being spoken of as myelomas, endotheliomas, angiomas, giant-celled tumors, xanthomas, or combinations of these terms.

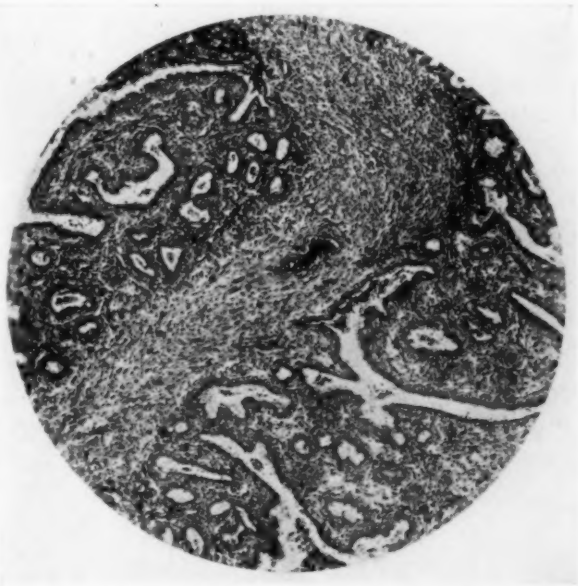

FIG. 7.—Cystosarcoma-phylloides, with xanthomatous degeneration of the connective tissue stroma. Lower power view, Case II. (Magnification 70 diameters.)

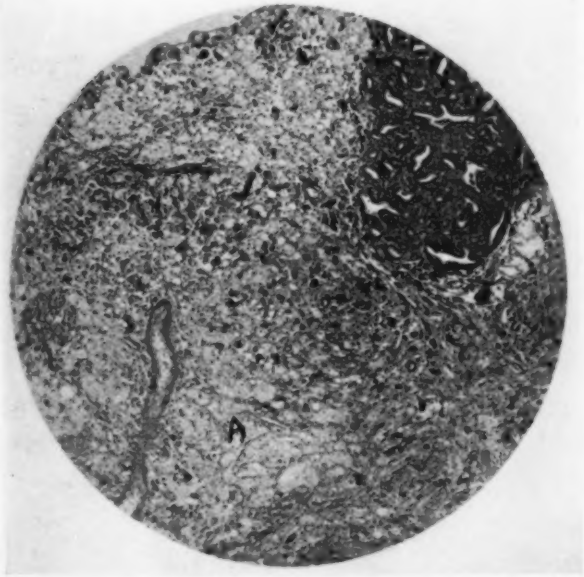

FIG. 8.—Cystosarcoma-phylloides, Case II. Low power showing groups of "foam" cells, A.

As far back as 1845 Lebert in his "physiologic pathologique" in describing certain fibro-plastique tumors speaks of their yellow color and the presence within them of a peculiar fat which he called "xanthos." Sir James Paget[25] in 1856 in his "Surgical Pathology" refers to myeloid tumors of the mammary gland, eyelids, conjunctiva, subcutaneous tissues and bone, in which he found large multinucleated giant cells. Heurtaux[26] of Nantes, in a classical monograph in 1891 first described myelomas of the tendon sheaths, report-

ing three cases, and classed them with benign tumors. All three presented a yellow color in certain areas and within the tissues an ether soluble fatty substance was found. Dor[27] in 1898 called them mycloxanthomas and expressed the belief that the xanthoma cells and the multinuclear giant cells had the same origin. Bellamy[28] in 1901 classed them as endotheliomas, inasmuch as in the evolution of the tumor the primary phase is a proliferation of the endothelial cells of the blood-vessels. Bloodgood[29] in 1903 and again in 1905[30] classified them among the benign hæmangiomas. Tourneux[31] in 1913 collected from the literature and from personal observation 93 cases of what he called xanthosarcomas of the tendon sheaths in which 54 were of the xanthoma type. The finger was the site of the lesion in 48, the hand in 12, forearm in 6, foot in 19, and the leg in 1. He states that they may grow very slowly and be stationary for years, and then suddenly grow rapidly, or may be malignant from the start. Those in the fingers seem to be most benign. In 21 there was recurrence after local removal. Landois and Reid[32] in 1915 described the foam cells and giant cells in a xanthoma which had been mistaken for a sarcoma, and expressed the belief that they were not pathognomonic of xanthoma and were due to absorption of cholesterin from the broken-down blood cells. Broders[33] of the Mayo Clinic in 1919 reported 17 cases of benign xanthic extra-periosteal tumors of the extremities of which 16 were connected with a tendon sheath. Foreign body giant cells and blood pigment were present in all of his cases and foam cells in 64 per cent. In 35.3 per cent. the etiology was associated with a definite history of injury. Ollerenshaw[34] in 1922 described bilateral symmetrical tumors of the Achilles tendon the size of walnuts which were removed at operation. They contained many giant cells and foam cells and he called them granulomas. In addition to the local tumors cutaneous xanthomatous lesions existed on the arms. Fleissig[35] in 1922, after reporting 3 cases of his own and reviewing many cases from the literature came to the conclusion that the so-called "myelomas" or "giant-celled sarcomas" of the tendon sheaths of the fingers, toes, hands and feet, forearms and legs, are not true tumors at all, but tendon sheath granulomas. They are all somewhat lobulated, have a yellow-red mottled color, and are of a firm elastic consistency. Microscopically there is a conspicuous absence of sarcoma tissue, a predominance of foreign body giant cells, and the blood pigment present in the tissue has nothing to do with the color of the tumor. They are benign and local removal is sufficient.

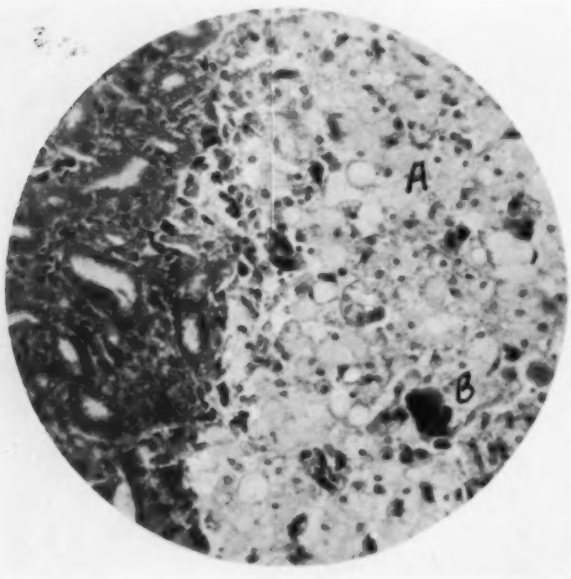

FIG. 9.—Case II. Cystosarcoma-phylloides. Higher power, showing "foam" cells, A and a foreign body giant cell, B. (Magnification 240 diameters.)

These views correspond in the main with those of Garrett,[36] who working in Bloodgood's laboratory, has very recently published a careful study of 196 soft tissue tumors. These included 76 fibrohæmangiomas, 30 fibromas of the tendon sheaths, 26

granulation tissue tumors, 53 cases of bursitis, and 11 tumors connected with joints. In this series the characteristic elements of xanthoma tissue, namely the yellow color, the foam cells, and the foreign body giant cells, could be made out in 19 of the fibro-hæmangiomas, in 11 of the tendon sheath fibromas, in one granulation tissue tumor, in 2 bursitis cases, and in 4 tumors connected with joints. The author differs with Fleissig in believing that the yellow color of these tumors is due entirely to pigment absorbed from the degenerated blood, lays particular stress upon their benign character, but at the same time strongly emphasizes the importance of thorough local removal.

While it is true that the majority of xanthomatous tumors are seen about the hands and feet, especially in connection with the tendon sheaths, other parts of the body are not infrequently involved. Thus Carroll Smith [37] in describing what he calls "true xanthoma tumors," reports one situated on the tongue, one on the labia majora involving the skin and submucosa, a third in the parotid gland and a fourth, a walnut sized tumor, in the dura over the region of the frontal lobe. All of these were benign tumors and were classed as xanthomatous endotheliomas. Of even greater interest is the report of Ewing [38] who in 1922 described typical xanthomatous tissue in connection with tumors of bone. "Some of the central giant-cell tumors of bone" he says, "are solid throughout, firm, dry and yellow. The yellow color is due to the presence of many large polyhedral cells distended with lipoid granules. These tumors have greater growth capacity than the ordinary giant-cell tumor. They often reach considerable size, breaking down the bony capsule and forcing their way between muscle and fascia, but definite infiltrative growth is rare. . . . Their structure presents a diffuse growth of medium sized spindle cells with very little hyperchromatism. Varying proportions of the tissue are composed of the lipoid cells. Giant cells are missing over large portions of the growth, but usually they appear abundantly in some portions, generally about blood spaces or blood extravasations. If the histologist encounters only the spindle cells, an erroneous impression of a malignant tumor is liable to be gained. . . . I have never known these tumors to produce metastases and I have been unable to find authentic records of such complication, but it seems quite possible that by curettage, groups of viable cells

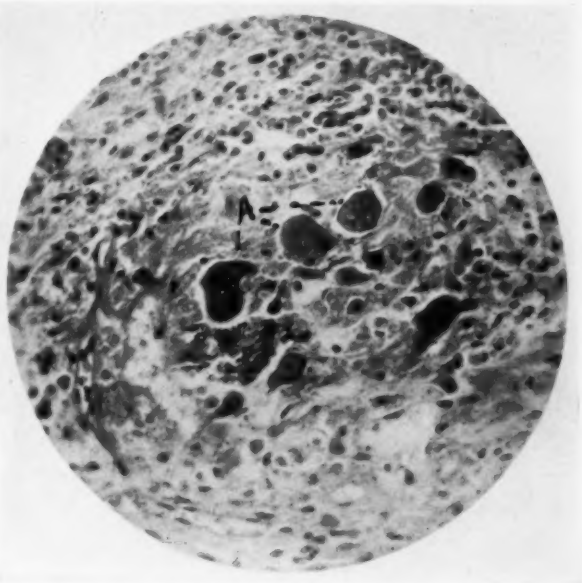

FIG. 10.—Case II, cystosarcoma-phylloides. Higher power, showing a group of multinuclear foreign body giant cells, A. (Magnification 240 diameters.)

could be dislodged from the tumor mass and pass into the blood-vessels. However, the blood-vessels are usually small and scanty and the danger of metastases can probably be ignored. That a central xanthosarcoma of bone is a variant of the giant-cell tumor is clearly indicated by the clinical history, central location, general structure, the course of the growth as revealed by the X-ray, and by the not infrequent occurrence of xanthoma cells in typical giant-cell tumors."

One might go on indefinitely and cite reports from other authors, without adding materially to the evidence already at hand that xanthomatous changes being of a degenerative character may occur in any type of endothelial or connective-tissue tumor in which the metabolic disturbances are favorable for their formation, and that by far the majority of tumors in which xanthomatous tissue is seen are benign in character and should be so surgically treated.

FIG. 11.—Multiple myelomas. At A, tissue was removed for microscopic section and showed typical xanthoma cells.

The author desires to present three clinical cases in which xanthomatous changes are well illustrated. The *first* is that of a Jewish woman about fifty years of age who came to the hospital because of tumor of the right foot which had been developing for the past two years. When first noticed it was a small pea-sized nodule on the dorsum of the foot; it grew rapidly to the size of a cherry and then very slowly enlarged until it has now become the size of a walnut. There was no injury associated with its onset, it was at no time painful, and the only reason for her coming to the doctor was the inconvenience it gave her in walking. On examination we found a non-inflammatory rather firmly fixed solid tumor the size and shape of half an egg with its broad base occupying the area over the web between the great and second toes. Its surface was smooth and the skin over it could be wrinkled between the fingers. X-ray showed no calcification and no bony abnormalities and we made a diagnosis of a benign, well encapsulated connective tissue tumor, probably a fibroma. Under local anæsthesia a longitudinal incision was made over the tumor and a definite line of cleavage followed between the skin and the fibrous capsule of the tumor. The separation was continued without difficulty down to the web between the toes where an indefinite connection with the sheath of the flexor tendons was divided and the utmor removed intact. On cutting through the tumor with a knife the tissue though fairly firm had an elastic spongy consistency, and the cut surface was of a diffuse brilliant yellow-orange color mottled with small areas of reddish-brown. (Fig. 1.) Microscopic examination (Fig. 2) showed a ground work of loose fibrous connective tissue rich in blood-vessels with clusters of

large polyhedral cells scattered diffusely throughout the tumor. Many large multinuclear foreign body giant cells were seen, and not a little yellow pigment. Under high power (Fig. 3) these large polyhedral cells were seen to have rather small dark nuclei, and the cytoplasm to be filled with fine granules which gave a yellow-red color with sudan III. and a gray color with osmic acid, the typical picture of the foam cells characteristic of xanthoma tumors. Repair of the wound was rapid and there has been no evidence of recurrence 6 months after operation (Fig. 4).

The *second* case is that of a woman of forty years, who entered the service of Doctor Lewis because of a tumor of the right breast which had been gradually developing over a period of 6 years, not associated with pain, glandular enlargement or discharge from the nipple. (Fig. 5.) Examination showed a tumor mass about the size and shape of a grape fruit occupying the anterior part of the breast. It had a peculiar violaceous color, was solid in consistency, presented a smooth surface and was freely movable over the pectoral muscles. A diagnosis of cystosarcoma-phylloides was made and this was confirmed by operation which consisted of a complete mammectomy. On cross-section of the tumor (Fig. 6) an area about the size of a hen's egg had a brilliant yellow-orange color which was in marked contrast to the remaining tissue. On microscopic section (Figs. 7, 8, 9 and 10) through this area the connective-tissue stroma of the long tree-like processes showed a large amount of blood pigment, many large multinuclear foreign body giant cells, and the typical foam cells of xanthoma tissue. This is a very unusual finding in a breast tumor and without doubt represents a degenerative process in the connective tissue in a rather limited portion of a large benign tumor.

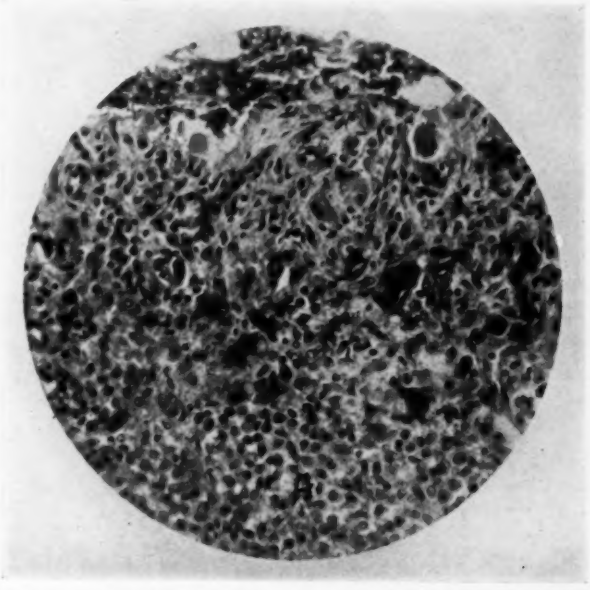

FIG. 12.—Multiple myelomas, Case III. Showing myeloid cells A and multinuclear giant cells B. (Magnification 250 diameters.)

The *third* case is that of a small boy who entered the service of Doctor Phemister with multiple myelomas involving the bones of the cranium, vertebræ and extremities. (Fig. 11.) For the purpose of microscopic study a piece of tissue was removed from a lesion in the shaft of the humerus. The predominating type of cells was the plasma cells and myelocytes, but in addition there were areas near the cortical bone in which large multinuclear foreign body giant cells were seen (Fig. 12) and in a few places the typical foam cells characteristic of xanthoma tissue were present (Fig. 13).

While these clinical reports add weight to the already well established views regarding the nature of xanthomatous tissue, there still remains the much disputed question as to what the yellow color of this tissue is due. Bloodgood and his students have attributed it to the presence of blood pigment, while Fleissig has emphatically expressed his belief that this is a negligible factor and that the real cause is the presence in the foam cells of the lipoid granules.

The ideas of most other writers have coincided with one or the other of these two views. The true solution in all probability lies between the two, that is, a combination of both. Prof. Le Roy Palmer [39] of the University of Minnesota has during the past few years made exhaustive studies of the pigments in plant and animal life, and certain facts established by him have a direct bearing on the point under discussion. He has shown that the orange, red and yellow pigments found throughout plant and animal life are the carotinoids, carotin and xanthophyll. Certain animals, such as the swine, sheep, goats, dogs, cats and guinea pigs, whose body fat is white, are devoid of these pigments in their blood. The carotin of butter fat, adipose tissue and blood serum of cattle is biologically derived from the food and that a similar relationship exists between xanthophyll of egg yolk and fowl tissue fat and plant xanthophyll. The pigments of human milk fat and probably human tissues in general very likely contain either of these pigments or both, and that there is evidence of a carotin albumen complex in the blood serum which may play an important part in the process of fat synthesis.

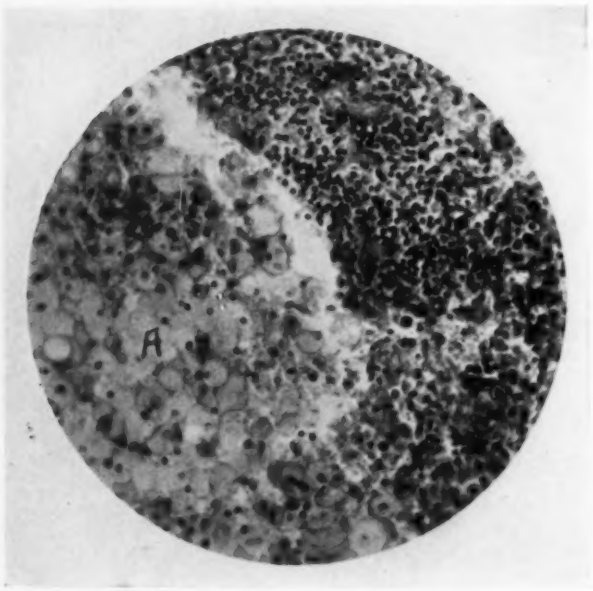

FIG. 13.—Case III. Multiple myelomas. Showing group of xanthoma "foam" cells A. (Magnification 250 diameters.)

In the light of these facts there is little doubt but that the brilliant orange-yellow color of xanthoma tissue is directly due to the presence of one or both of these pigments, which being normally present in the blood serum are, as pointed out by Wells, readily taken up and combined with the cholesteral fatty acid esters (themselves colorless) which in all xanthoma tissues are so abundantly present.

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EFFECTS OF THE INTRAMUSCULAR INJECTION OF SODIUM CITRATE UPON BLEEDING*

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Historical Review.— In 1915, sodium citrate was first used clinically to prevent coagulation of blood in transfusion operations. Being recognized as an anticoagulant, the question logically arose: would the introduction of the citrate suspend coagulation of the recipient's blood? This did not occur, as was soon established: in fact only a transient shortening of the coagulation time of the recipient was found to follow transfusion of citrated blood. Neuhof carried out some experiments along this line in 1916 consisting of the intravenous injections of solutions of sodium citrate into dogs under ether anæsthesia. The following results are quoted:[1]

"(1) The coagulation time is tremendously shortened within a few minutes after the introduction of non-toxic doses of sodium citrate, and this shortened coagulation may be sustained for one or more days; (2) The bleeding time is likewise shortened so that, after citrate injection, a large vessel can be incised with prompt coagulation about the wound; (3) Coincident with the shortened coagulation time, the color of the venous blood is altered to a light arterial tint. (4) There is no fixed toxic or lethal dose of sodium citrate per kilo of body weight, toxicity depending to a remarkable degree upon the rate of introduction of the citrate solution. (5) A toxic or lethal dose is characterized by a swing from the state of shortened coagulation to one of suspended coagulation. This latter phenomenon led to the sodium citrate method of blood transfusion, and it was this effect which overshadowed the ordinary pharmacological action of sodium citrate."

Observations on the intravenous injection of sodium citrate on the human being have been few and far between. Weil[2] in 1915, reported shortening the coagulation time one-half by the administration of 5 gms. of a 20 per cent. solution in an adult. In 1916, Ottenberg[3] described a case of hæmophilia in which 6 gms. caused a marked drop in the coagulation time with a prolongation of the coagulation time 48 hours later. Kinsella and Brown[4] in 1920, reported an attempt to control pulmonary hemorrhage during the course of influenza by the intravenous injection of sodium citrate in one gram doses and described the effect on the coagulation time in five patients.

By far, the most exhaustive and complete work on this subject up to date has been that of Neuhof and Hirschfeld.[5] They report a series of 500 cases to whom sodium citrate has been administrated, the last 200 being by the intramuscular route and conclude that such administration results in prompt

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and pronounced shortening of coagulation and bleeding time which is of two or three hours duration with a gradual return to normal within twenty-four to forty-eight hours. They established the optimum dose as 30 c.c. of a 30 per cent. solution, and the intramuscluar route as the one of choice.

Our report is based upon clinical observations made in a series of over fifty cases to whom sodium citrate was administrated intramuscularly. For several years the writers have independently used calcium lactate preparatory to operations, especially before tonsil operations. While no deleterious effects were observed, the clinical effects appear to be negative. With the advent of World War Veterans to the surgical service of the Soldiers Home in Milwaukee it has been the rule, particularly for ear, nose and throat patients, to admit them the day before the operation, the preparatory examination to include the determination of the patient's coagulation and bleeding time. The observations on the first patient of this series are typical.

Effects of the Administration of Sodium Citrate.—The coagulation time of this patient was eleven minutes on admission; ten minutes following the injection the coagulation time dropped to nine minutes; twenty minutes after injection the time was down to seven minutes; thirty minutes after injection the time was down to four minutes; forty-five minutes following injection, the lowest point was reached in this case, being two minutes. From then on there was a return to normal, but more gradual than the abrupt fall. One hour after injection the coagulation time in this case was three minutes; six hours after injection it was six minutes; twelve hours after injection it was seven minutes; eighteen hours after injection it was nine minutes; and until the coagulation time previous to the injection was reached. The field was drier following tonsillectomy in this case than the usual non-citrated case with normal coagulation time. This same patient was operated seven days later for deviated septum. His coagulation time then was nine minutes. The accompanying chart on the first twenty-five cases is of interest and speaks for itself. If necessary, the dose can be repeated in thirty-six hours, thereby controlling the coagulation time for a longer period. The majority of our cases were patients on whom nose and throat operations were performed and the results were highly gratifying. Any operator with much experience in tonsil surgery knows he must guard against hemorrhage. In all our cases of tonsillectomy previously citrated the field of operation was dry and remained so, and rarely sutures were necessary. In view of one septum patient who had some bleeding and the pleasing result of the other patients injected before operation, it is our opinion that sodium citrate is more effective when administrated before the operation. If morphine or another narcotic is employed before operation the citrate can be conveniently injected at that time. That it is of some value when used during an operation is attested by its administration given during the course of a radical mastoid operation in a case of unusual, though not serious, oozing from the mastoid

bone. In this instance there was less sponging required than is usual when completing the operation some thirty minutes later in the attic and middle ear. In a case of common duct obstruction due to cholelithiasis, accompanied by intense jaundice, the coagulation time was reduced from nine to two minutes one hour after citrate injection, and no difficulty with bleeding was encountered during or after the operation. Sodium citrate was injected intramuscularly in three cases of pulmonary hemorrhage due to pulmonary tuberculosis, and while it cannot be conclusively stated that this was the sole factor in checking the hemorrhage; nevertheless it can be said that bleeding ceased twenty minutes following the injection. One case of intra-abdominal hemorrhage due to perforation of the cæcum was successfully controlled by this method. No untoward results, other than that of temporary local pain, have occurred in any of the injections.

Nature of the Reaction.—Baer and Rosenthal[6] in a personal communication to Neuhof and Hirschfeld reported the results of their studies on citrated blood and concluded that the action of the sodium citrate was directly upon the blood platelets destroying them and liberating a substance which activates the process of coagulation. Drinker and Brittingham[7] have demonstrated changes in the blood platelets accompanying early coagulation. Many of the platelets are partially or totally destroyed in transfusion of citrated blood. Hence in any disease such as hæmophilia, purpura hemorrhagica, pernicious anemia or Banti's disease where the pathology is a marked diminution or absence of blood platelets the injection of sodium citrate should not be used and in fact would tend to have a deleterious effect. This result is reported in Ottenberg's case of hæmophilia which gave a slight primary drop in the coagulation time, but soon showed a marked prolongation far beyond the original coagulation time.

Technic.—The solution used is sodium citrate C. P. 30 per cent. sterilized by boiling. The dose given is 30 c.c. and the site of injection is the buttocks. The skin over each buttock is cleansed with alcohol, dryed, and tincture of iodine applied over a broad area. With a three inch needle, 3 c.c. of a 1 per cent. novocain solution is injected into each buttock. Three minutes are allowed to elapse, and then 15 c.c. of the citrate solution are injected into each buttock directly in the same area as in the injection of novocain. The novocain tends to diminish to a great degree the pain, and we find that gentle manual massage likewise alleviates the soreness which most of the patients complain of, but which is temporary only. Where time is an important factor, or where the condition of the patient is such that he cannot be moved, the entire 30 c.c. may be given in one buttock. This was done in the cases of the pulmonary † and intra-abdominal hemorrhages and mastoiditis with no untoward results. Tenderness was noticed in a few instances, but this was relieved even in the sitting position by a soft pillow or rubber

† Dr. Dunham of Oak Forest Sanatorium, Oak Forest, Illinois has successfully checked three cases of pulmonary hemorrhage by this method. Personal communication to the authors.

TABL

Table Showing Results in th

Case number	1	2	3	4	5	6	7	8	9
Coagulated time before citrate	11 min.	9 min.	8 min.	6 min.	9 min.	10 min.	7 min.	8 min.	10 min
10 minutes following injection	9 min.	9 min.	7 min.	6 min.	8 min.	9 min.	6 min.	7 min.	8 min
20 minutes following injection	7 min.	7 min.	7 min.	4 min.	6 min.	7 min.	4 min.	4 min.	6 min
30 minutes following injection	4 min.	5 min.	4 min.	2 min.	4 min.	4 min.	2 min.	3 min.	5 min
45 minutes following injection	2 min.	2 min.	1½ min.	1 min.	2½ min.	2 min.	1½ min.	2½ min.	5 min
60 minutes following injection	3 min.	2 min.	3 min.	3 min.	3 min.	4 min.	2 min.	2½ min.	2 min
6 hours following injection	6 min.	3 min.	3 min.	3 min.	4 min.	5 min.	4 min.	4 min.	5 min
12 hours following injection	7 min.	5 min.	5 min.	4 min.	4 min.	5 min.	4 min.	4 min.	7 min
18 hours following injection	9 min.	6 min.	5 min.	4½ min.	5 min.	7 min.	5 min.	5 min.	7 min
24 hours following injection	9 min.	6 min.	7 min.	5 min.	7 min.	7 min.	5 min.	5 min.	9 min
36 hours following injection	10 min.	9 min.	8 min.	5 min.	9 min.	9 min.	6 min.	8 min.	10 min
48 hours following injection	10 min.	9 min.	8 min.	5 min.	9 min.	9 min.	7 min.	8 min.	11 min

TABLE I.

Results in the First Twenty-five Cases

	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
nin.	10 min.	9 min.	7 min.	6 min.	8 min.	10 min.	9 min.	7 min.	8 min.	9 min.	6 min.	8 min.	5 min.	9 min.	9 min.	7 min.	6 min.
nin.	8 min.	7 min.	5 min.	4 min.	7 min.	8 min.	7 min.	4 min.	7 min.	7 min.	4 min.	6 min.	3 min.	7 min.	8 min.	6 min.	4 min.
min.	6 min.	7 min.	4 min.	3 min.	5 min.	4 min.	5 min.	4 min.	5 min.	5 min.	2 min.	5 min.	3 min.	5 min.	6 min.	4 min.	4 min.
nin.	5 min.	4 min.	2 min.	1 min.	4 min.	3 min.	5 min.	2 min.	3 min.	3 min.	2 min.	3 min.	2 min.	3 min.	4 min.	4 min.	3 min.
nin.	5 min.	4 min.	2 min.	1 min.	2 min.	3 min.	2 min.	1 min.	2 min.	2 min.	1 min.	3 min.	1 min.	2½ min.	3 min.	2 min.	2 min.
nin.	2 min.	1 min.	1 min.	2 min.	2 min.	2 min.	3 min.	1 min.	2 min.	2 min.	2 min.	2 min.	1 min.	3 min.	2 min.	1 min.	1 min.
min.	5 min.	2 min.	4 min.	4 min.	4 min.	4 min.	5 min.	3 min.	4 min.	4 min.	4 min.	4 min.	2 min.	4 min.	2 min.	2 min.	2 min.
min.	7 min.	5 min.	4 min.	5 min.	5 min.	6 min.	7 min.	4 min.	5 min.	7 min.	4 min.	6 min.	4 min.	4 min.	4 min.	4 min.	4 min.
min.	7 min.	7 min.	6 min.	5 min.	7 min.	8 min.	9 min.	6 min.	6 min.	7 min.	5 min.	7 min.	4 min.	6 min.	7 min.	5 min.	5 min.
min.	9 min.	8 min.	7 min.	6 min.	7 min.	9 min.	9 min.	7 min.	8 min.	8 min.	6 min.	8 min.	5 min.	7 min.	8 min.	6 min.	6 min.
nin.	10 min.	9 min.	8 min.	6 min.	8 min.	10 min.	10 min.	7 min.	8 min.	9 min.	6 min.	8 min.	5 min.	9 min.	9 min.	7 min.	6 min.
nin.	11 min.	9 min.	8 min.	6 min.	8 min.	10 min.	10 min.	7 min.	9 min.	9 min.	6 min.	8 min.	5 min.	9 min.	9 min.	7 min.	7 min.

ring. One case was followed by marked infiltration of one buttock which absorbed without fluctuation, with the aid of hot compresses; the pathology in our opinion being hæmatoma. Citrate was used by one of us in consultation in a severe case of post-operative hemorrhage in which horse serum and similar agents were administered before the citrate injection.

SUMMARY

1. The intramuscular injection of sodium citrate causes a prompt reduction of the coagulation time of a duration of one to three hours with a gradual return to normal with twenty-four to forty-eight hours.

2. This does not occur when the pathology is marked diminution or absence of blood platelets as in purpura, hæmophelia, etc.

3. A 30 per cent. C. P. sodium citrate solution is used, 15 c.c. in each buttock preceded by the injection of novocain. This method is apparently free from danger, no untoward results having been noted in over fifty cases.

4. The citrate cure occurs not only in persons who have an average coagulation time, but also in those having an abnormal prolongation, as in jaundice or from unknown causes.

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INTRACRANIAL ARTERIO-VENOUS ANEURISM OR PULSATING EXOPHTHALMOS

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(continued from p. 24)

CASE REPORTS COLLECTED BY THE AUTHOR

SPONTANEOUS TYPE

(1) Bruce.—Rev. Neur. and Psychiat., Edin., 1908, vol. vi, p. 462. F. 74, O.S. Spont. —sudden onset of pain all over head and subj. bruit—swelling of lids of both eyes—1 month later puls. exoph. O.S. with chemosis—fundus O.S. shows only congestion of veins—bruit systolic in time and widely transmitted—ext. ophthalmoplegia with ptosis—pupil somewhat dil. and reactionless—(V) impaired—abducens paresis O.D.—(T) conservative with rest, KI and sedatives—death—no autopsy.

(2) Bourget and Mireaux.—Toulouse Méd. 1909. vol. ix, p. 104. F. 28, O.S. Spont. Young woman with goitre noticed œdema of l. side of face and diminishment of (V) O.S.—puls. exoph. with subj. and obj. bruit—chemosis and dil. of veins of eyelids and conj.—fundus O.S. shows swollen veins—pupil dil. and reacts sluggishly to l.—no Wassermann nor X-ray (T) medication and digital compression for 5 days without result —lig. of carotid 3 months after onset produced considerable improvement.

(3) Fry.—St. Barth. Hosp. Jour., 1910, vol. xviii, p. 40. F. 31, O.D. Spont. Suddenly while working had sharp pain of rt. temple—2 days later bruit and in 5 days diplopia and int. strabismus of O.D.—puls. exoph.—puls. swelling and thrill of brow—dil. veins of conj. and sclera—fundi O.D. and O.S. = veins full—(V) O.D. = 6/9; O.S. = 6/6—visual fields neg.—urine Sp. G. 1026, alb. neg.—compression of carotid stopped bruit —(T) digital compression will be used.

(4) Albertin and Desgouttes.—Recueil d'Ophth., 1910, vol. xxxii, p. 31. Sex ?, age ?, O.S. Spont. Sudden onset of puls. exoph.—in a few days amaurosis of O.S.—chemosis —lig. of left common carotid caused sl. hæmiparesis which cleared in 3½ days—puls. exoph. was cured but there was some loss of (V) from opacities.

(5) Moreton.—St. Barth. Hosp. Rep., London, 1914, vol. xl, p. 73. F. 32, Side ?. Spont. onset with severe pain across temple and subj. bruit—typical puls. exoph. developed —no Wassermann nor X-ray—(T) systematic digital compression of carotid without avail.

(6) Vail and Oliver.—Lancet Clinics, Cincinnati, 1914, vol. cxii, p. 644. F. 72. O.S. Spont. (6 months previous to onset had rather insignificant trauma to l. frontal region)—onset at night—awakened by bruit and pain of O.S.—puls. exoph. with bruit best heard over O.S.—distinct pulsation near inner canthus—tenometer readings O.S. = 65 mm., O.D. = 20 mm.—complete ophthalmoplegia ext.—pupil dil. but reacts feebly to l. = V. O.S. = 20/50, O.D. = 20/20—visual fields neg.—fundus O.S. ? because of hazy media —O.D. shows pulsation of arteries and veins—B.P. 200/72—no Wassermann nor X-ray—compression of l. carotid shuts off bruit and pulsation (T) lig. of l. common carotid resulted in remarkable cure.

(7) Snowball.—Trans. Ophth. Soc., United Kingdom, London, 1914–15, vol. xxxv, p. 169. M. 13, O.D. Spont. onset with intense headaches and vomiting—puls. exoph. with chemosis and widely transmitted bruit—dil. of vein about orbit with thrill—fundi O.D. shows retinal veins full and haziness of disc.—O.S. sl. swelling of upper inner

Abbreviations: M—Male; F—Female; arabic numerals indicate age of patient; V—Vision or Visual acuity; T—Treatment; Dil.—Dilated or Dilatation; Lig.—Ligation; Conj.—Conjunctivum; Sl.—Slight; Puls.—Pulsating or Pulsation; Obj.—Objective; Subj.—Subjective; Dim.—Diminished; O.D.—Right eye; O.S.—Left eye; Bilat.—Bilateral.

margin of disc—movements of O.D. restricted—pupils equal not dil. but O.D. reacts only sl.—no Wassermann—X-ray neg.—pressure over carotid stops bruit—medication by mouth of no avail but no preliminary course of digital compression of carotid (T) lig. of rt. carotid and ext. jugular, facial and supraorbital veins improved puls. exoph. but death occurred in 9 days. Autopsy—arterio-venous communication between cavernous sinus and int. carotid.

(8) DAMSGAARD.—Ugeskrift f. laeger, 1915, vol. lxxvii, p. 20. F. 78, Bilat. Spont. onset with violent pain of l. side of head especially in the temple with subj. and obj. bruit—in a few days exoph. appeared with proptosis and chemosis—23 days after onset pulsation of O.S. noticed—extraocular palsies which increased to complete ext. ophthalmoplegia—almost absent papillary reaction—no Wassermann, no X-ray—no history of lues—compression of l. carotid stops bruit—rt. does not—lig. of l. int. carotid without preliminary digital compression caused bruit to cease but hæmiplegia resulted, stupor and death 2 days later—no autopsy.

(9) KRAUSE.—Trans. Col. Phys., Philadelphia, 1915, 3. a., vol. xxxvii, p. 467. F. 57, Bilat. Spont. enormous bilat. exoph. O.D. more than O.S. which subsided for a time and then returned—continuous bruit accentuated in systole heard all over head, loudest over O.D.—pulsation of O.D. very marked—bilat. proptosis—complete paralysis of ocular muscles of O.D. with dil. and immobile pupil and complete blindness—fundi showed O.D. dil. veins, retinal hemorrhages with œdema of nerve and retina—later synechia and vtireous opacities O.S. vessels full, no neuritis—bruit ceases with compression of carotid—B.P. 202/125—Wassermann, blood and spinal fluid neg.—X-ray of skull neg.—visual fields concentrically retracted—T. of rest and digital compression caused sl. improvement, then recurrence—more digital compression effected some improvement O.S. becoming normal except for atrophy of disc and diminishment of V. to 5/18.

(10) TINDALL.—Hahnemann Monthly, Philadelphia, 1915, vol. l, p. 671. F. 40, O.D. Spont. with onset of pain in globe and noise in rt. ear—puls. exoph. with bruit—congestion of conj. vessels—fundus O.D. showed questionable pallor of disc—diplopia and marked involvement of extraocular muscles—V. dim.—Wassermann negative—X-ray unsatisfactory—T. by pressure bandages to eye, mercury and iodides for 6 wks. followed by improvement in exoph. and ext. ocular palsies.

(11) RISLEY.—Trans. Col. Phys., Philadelphia, 1916, 3. s., vol. xxxviii, p. 343. Discussion of de Schweinitz and Holloway's paper.) Sex ?, age ?, side ?. Spont. puls. exoph. with bruit and proptosis—compression of carotid stopped bruit—T. by intermittent digital compression of carotid following which bruit, pulsation, and proptosis disappeared.

(12) MORAX and DUCAMP.—Ann. d'Ocul., Paris, 1916, vol. cliii, p. 252. F. 68, O.D. Spont. puls. exoph. with bruit over rt. temporal region accentuated during systole—fundus neg.—ptosis O.D.—diplopia—paresis of the extraocular muscles—pupil O.D. dil. and sluggish reaction—V. = 5/15—visual fields normal—no X-ray nor Wassermann—subcutaneous injection of gelatin without avail—lig. of rt. int. carotid without preliminary digital compression—6 days later l. hæmiplegia, exoph. and pulsation gone—death 36 days after operation. Autopsy—communication between int. carotid and cavernous sinus.

(13) FISHER.—Proc. Roy. Soc. Med., 1916, vol. ix, Part 3, Sec. of Ophth., p. 24. F. 59, O.D. Spont. onset with dizziness then fell unconscious—vomiting and pain in rt. frontal region—puls. exoph. O.D.—obj. bruit heard over eye and in fronto-temporal region—veins of upper lid distended—fundus O.S. showed veins fuller than O.D.—ophthalmoplegia ext. and fixed pupil—V. = 6/14 corrected—Wassermann neg.—X-ray neg.—T. -Kl administered by mouth, sl. improvement in sight and in eye movements.

(14) WATANABES.—Nippon Gank. Zasshi, July, 1916, p. 175. M. 19, side ?. Spont. puls. exoph.—good results followed lig. of the common carotid.

(15) RIDLEY (Discussion of Mayou's article, 1917, see below).—Sex ?, age ?, side ?. Spont. puls. exoph. and proptosis that was thought to be due to gumma—lig. of common carotid without benefit—dil. of facial artery on same side—after lig. of facial artery puls. exoph. cured but blindness of l. eye.

(16) FRITSCH.—Ztschr. f. Augenh., 1917, vol. xxxviii, p. 186. F. 60, O.S. Spont. Since childhood had prominence of and diminished V. in O.S.—noise in head, and headache— puls. exoph.—bruit subj. and obj. heard over O.S. and l. side of head—chemosis —fundus shows dil. veins and atrophy of disc—tension O.S. greater than O.D.—int. strabismus O.S. and reactionless pupil—no Wassermann nor X-ray—double lig. of l. common carotid followed by improvement.

(17) BOOT.—Illinois Med. Jour., 1918, vol. xxxiv, p. 217. F. 21, O.D. Spont.—2 years ago noise in rt. ear and 9 months ago O.D. injected and swollen—now exoph. 3 mm., chemosis and bruit widely transmitted—large dil. veins of upper lid and inner angle of orbit with thrill—fundus O.D. marked fulness of veins—no mention of cranial nerve involvement—no Wassermann or X-ray—lig. of rt. carotid followed by complete cure.

(18) ZEEMAN.—Nederl. Tijdschr., von Geneesk, Amsterdam, 1918, vol. i, p. 778. F. 53, O.S. Spont. puls. exoph. 7½ mm.—pupil O.S. dil. with no reaction to l.—fundus O.S. shows pale disc—absolute blindness O.S.—Wassermann positive—anti-luetic without results—lig. of ophthalmic artery and at which time a soft pulsating tumor of orbit was seen—sl. improvement, then recurrence and death. Autopsy, no details except multiple myelomata of orbit.

(19) TYSON.—Arch. of Ophth., N. Y., 1919, vol. xlviii, p. 523. F. 43, O.S. Spont. with onset of severe pain behind O.S. and subj. bruit—puls. exoph. with obj. bruit and dil. of veins about eye—fundus shows œdema of nerve head and retinal hemorrhages—diplopia, pupil dil. with sluggish reactions and ext. ophthalmoplegia—7th nerve palsy—(V) O.D. = 20/30; O.S. = 20/50 corrected—X-ray neg.—Wassermann neg.—(T) by digital compression and iodides with resulting cure.

(20) LIEGARD.—Ann. d'Ocul., Paris, 1919, vol. clvi, p. 621. F., age ?, bilat. Spont. onset with headache and vomiting—bruit in l. temporo-parietal region—bilat. exoph. without visible pulsation—paralysis of l. 3rd nerve—(T) by gelatin injections—end results were disappearance of exoph. and bruit but there was a 5th nerve palsy and loss of (V) of O.D.

(21) RUTTIN.—Wien. Med. Wchnschr., 1920, vol. lxx, p. 2027. F. 73, O.S. Spont. exoph. O.S. 4 mm.—systolic bruit loudest over l. temple—dil. of veins of inner canthus and episcleral veins—l. 6th and 7th nerve palsy—pupil dil. and reacts sluggishly—tension high—(V) O.D. = 6/6, O.S. = 6/12—Wassermann neg.—no X-ray—compression of l. carotid diminishes bruit—no (T) reported.

(22) SATTLER.—Ztschr. f. Augenh., Berlin, 1920, vol. xliii, p. 534. F. 56, O.S. Spont. —after confinement 8 years ago developed intracranial bruit and headache—puls. exoph. 5 mm. with bruit accentuated in systole and heard best at inner canthus O.S.—swelling of upper lid—fundus O.S. sl. puls. of veins and paleness of disc—(V) O.S. = 5/30, O.D. = 5/5—sl. extraocular palsies—l. supraorbital nerve sensitive to pressure—Wassermann neg.—compression of carotid stops bruit—medication and digital compression resulted in improvement and later 6 subcut. gelatin injections gave further improvement.

(23) GOMES.—Brazil Med., Rio de Janeiro, 1920, vol. xxxiv, p. 29. Sex ?, age ?, bilat. Spont. puls. exoph. of both sides—lig. of one common carotid without results and 3 months later expects to lig. the opposite common carotid.

(24) DEMARIA and JORGE.—Rev. Assoc. Méd. Argent., 1921, vol. xxxiv, p. 29. F. 42, O.S. Spont.—Pt. arteriosclerotic with exoph. goitre—sudden onset with pain of O.S. and inc. of exoph. O.S. with puls.—loss of (V)—bruit—dil. of veins at inner angle of O.S. with thrill—dil. of conj. veins—sl. diminishment of ocular movements—tension 42 mm.—X-ray neg.—Wassermann neg.—later (V) gone—pupil fixed—compression of carotid diminished exoph. and bruit—gelatin injections, Hg. by mouth and digital compression used without avail—excision of dil. veins above eye—cure.

(25) BEHAN.—N. Y. State Jour. Med., 1921, vol. xxi, p. 373. M. 27, O.S. Spont. onset purplish color of lower lid and 2½ months later headache and bruit—puls. exoph.

8 mm. with obj. bruit—conj. and scleral vessels distended—prominent int. angular vein with thrill—fundus O.S. shows engorgement of vessels, haziness of nasal margin of disc—visual field O.S. shows marked restriction both nasal and temporal—sl. extraocular muscle palsies—pupil O.S. greater than O.D.—(V) poor O.S. but had been for 2 years—no Wassermann—no X-ray—pressure of l. carotid obliterates bruit—(T) by carotid lig. without preliminary course of digital compression—cure of puls. exoph. and visual field enlarged—sudden death 4 months later—no autopsy.

(26) HALLETT.—Am. Jour. Ophth., 1921, 3.S., vol. iv, p. 203. F. 74, O.S. Spont. puls. exoph. with subj. bruit, also obj. bruit, latter heard over orbit, l. frontal and temporal regions—proptosis—acute glaucoma chemosis, tension 68 mm.—dil. tortuous veins of upper lid and about orbit—complete ptosis O.S.—dil. reactionless pupil—ophthalmoscopic exam. showed central retinal atrophy from an old hemorrhage and vitreous opacities—no (V)—pressure over l. carotid or over angular vein caused bruit to subside—Pt. was suffering from nephritis—no Wassermann—no X-ray—lig. of l. common carotid and l. angular vein without preliminary treatment resulted in death—no autopsy.

(27) MÉRIGOT DE TREIGNY.—Arch. d'Ophth., 1921, vol. xxxviii, p. 568. F., age ?, Side ?. Spont. subj. bruit—exoph. but puls. not visible—œdema of lids—bruit obj. with thrill—complete ptosis—intermittent digital compression of carotid without avail—lig. of common carotid and orbital veins followed by improvement.

(28) WHEELER.—Arch. Ophth., 1922, vol. li, p. 401. F. 62, Bilat. Spont.—both moderate and excruciating pains of the l. side of head and subj. intracranial bruit—swelling and discoloration about orbit—bilat. puls. exoph. greater on l.—bruit heard all over head reduced by pressure over l. carotid—fundi neg.—B.P. 185/110—Wassermann neg.—X-ray suggested tumor?—lig. of l. carotid without preliminary (T)—hæmiplegia and death 39 hrs. later—no autopsy.

(29) BRYAN.—Am. Jour. Ophth., 1922, vol. v, p. 808. M. 5, Bilat. Spont.—Puls. exoph. with bruit heard all over head—distended veins of temple and upper and lower lids—bilat. choked disc—(V) O.S. = 15/15, and O.D. = 15/15—no extraocular muscle palsies—visual fields normal—Wassermann neg.—X-ray neg.—bruit not abolished when both carotids were compressed—KI by mouth gave no results—later lig. of l. carotid did not cause improvement—still later lig. of l. superior ophthalmic vein was of no avail.

(30) RIEHM.—Deut. Med. Wchnschr., 1922, vol. xlviii, p. 287. F. 43, Bilat.—Spont. —3 miscarriages but denies lues—in 1918, grippe and pain in O.S. and left ear—6 months later O.S. prominent and red, and pain commenced in O.D.—dil. puls. veins about eyes—then puls. exoph. of both eyes and bruit—fundi O.D. and O.S. = choked discs with hemorrhage of O.D.—rt. 6th nerve palsy—pupils react normally—(V) O.D. = 6/30; O.S. = 6/12 —Wassermann neg.—X-ray showed thinning in region of sella—compression of both carotid arteries stopped bruit while compression of one only diminished bruit—no (T)—hernia operation, at which time Tb. lesions were found—death 12 days later. Autopsy—communication between int. carotid and cavernous sinus and Tb. peritonitis and salpingitis.

(31) DEMELLO.—Arch. Brazil de Med., Rio de Janeiro, 1922, vol. xii, p. 143. F. 60, O.D., Spont. Ptosis of O.D. and immobile globe appeared 5 years ago after a fever at which time there was severe pain of rt. face and 5th nerve anæsthesia—puls. exoph.—bruit of rt. temp. region—(V) O.D. = 1/10, O.S. = 1/4.

(32) GAZEPIS.—Arch. f. Ophth., 1922. vol. cx, p. 375. Sex ?, age ?, O.S. Spont. with onset of 6th nerve paresis of O.S. and then exoph. without puls. but with bruit heard over temples—dil. of orbital blood-vessels and fundus O.S. showed dil. and tortuous veins—ocular tension 82 mm.—developed glaucoma of effected eye—general arteriosclerosis—lig. of left int. and ext. carotids without preliminary course of digital compression—exoph. relieved but hæmiplegia resulted and death in 3 days. Autopsy, aneurism of left carotid in cavernous sinus and softening of left cerebrum.

(33) CAUCHOIX.—Bull. et Mem. de la Soc. de Chir., 1922, vol. xlviii, p. 20. F. 75, O.D. Spont.—awoke with violent pain and throbbing of head and heard bruit—exoph. with widely spread obj. bruit—chemosis—swelling of eyelid with thrill over it—somno-

lence—spinal puncture showed bloody fluid—Wassermann on fluid neg.—compression of carotid stopped bruit but caused syncopy—attempted lig. of ophthalmic vein with so great hemorrhage that the operator found it necessary to lig. rt. common carotid—sl. paresis of left face after operation cleared quickly and eye signs disappeared—3 months later a left hæmiplegia.

(34) FISHER.—Proc. Roy. Soc. Med., vol. vi, Part 3, Sect. Ophth., May, 1923, p. 99. F. 54, O.S., Spont. After doing washing became dizzy and fell, striking the back of her head—on regaining consciousness heard bruit—next day ptosis of O.S.—8 days later 3rd nerve palsy and dil. pupil O.S.—puls. exoph. O.S. and loud systolic bruit heard over globe and left temporal and frontal region—fundus O.S. shows dil. veins—(V) O.S. = 6/18—X-ray neg.—no Wassermann—following lig. of common carotid pulsation and bruit disappeared—loss of vision O.S. to 6/24 and some limitation of movements of globe remained.

(35) WHITHAM.—Am. Jour. Ophth., 1923, vol. vi, p. 81. F. 26, O.S. Spont. appearing 10 days after parturition with subj. bruit and pain across forehead—œdema of lids and conj. of O.S.—puls. of globe but no definite exoph.—obj. bruit widely transmitted—fundus normal—complete ext. rectus paralysis O.S.—pupil normal (V) O.S. = 20/30 imperfectly—B.P. 140 systolic—Wassermann neg.—no X-ray—pressure over carotid stopped bruit—lig. of left int. carotid followed by cure.

(36) *Ibid.* F. 36, O.S., Spont. onset while recovering from miscarriage—ptosis and diplopia—int. convergence with left 6th nerve and partial left 3rd nerve palsy—1½ months later exoph. O.S. 7 mm. with loud and widely dispersed bruit—thrill—veins of lid O.S. dil. with marked œdema of bulbar conj.—fundus shows engorged veins without puls.—no Wassermann nor X-ray—pressure on carotid stops bruit—(T) by rest in bed with neg. results—died from influenza—no autopsy.

(37) *Ibid.*—M. 58, O.S. Spont. onset—exoph. 3–4 mm.—œdema of conj. with dil. veins—fundus shows deep physiological cup—V. = 20/30, partial ptosis—diplopia—paresis of ext. rectus O.S.—4 mos. later V. = 20/70, and complete 6th nerve paralysis and contraction of visual fields O.S.—4 mos. exoph. showed puls. for the first time—deafness left ear—obj. bruit—Wassermann neg.—X-ray neg.—pressure on left carotid stopped bruit—T. by iodides—condition unchanged.

(38) DAMEL and BARRIOS.—Rep. Ophth. Hosp., Buenos Aires, vol. i, p. 130. M. 61, Side ? Spont. onset with diplopia then facial paralysis and then puls. exoph.—loud intracranial bruit—no extraocular muscle palsies—V. good—compression of common carotid gave some relief of signs and symptoms.

TRAUMATIC TYPE

(1) CHEATHAM.—Louisville Monthly Jour. Med. and Surg., Jan., 1907, p. 267. M. 48, O.D. Fall followed by puls. exoph. with bruit over rt. temporal region—ptosis—fundus O.S. shows optic atrophy—(V) loss except for light perception—no Wassermann—no X-ray—lig. of rt. common carotid gave improvement for a time and then recurrence—neg. results.

(2) MENACHO.—(Discussion of Blanco's paper—see above). F. 57, Side ? Following trauma of the side of her head, pt. developed puls. exoph.—compressive dressing caused cure in 6 weeks.

(3) GIFFORD.—Ophthalmology, 1907, vol. iv, p. 21. M. 24, O.S. 6 months after head trauma developed puls. exoph. and 2 months later first noticed subj. bruit which could be best heard over O.S.—dil. of veins about eye—fundus normal—(V) O.S. = 20/50—lig. of ext. carotid gave neg. results—later lig. of common carotid and dil. of upper lid and still progression in signs—(V) diminished and puls. tumor with thrill and bruit appeared about eye—limitation of ocular movements—removal of mass relieved exoph.—improved ocular movements—(V) regained to 20/30—fundus showed sl. pale disc.

(4) DESCARPENTRIES.—Bull. de la Soc. de med. du Nord, 1908, Lille, 1909, pp. 501–503, De Raffele. M. 25, O.D., Trauma with loss of consciousness and ecchymosis about

O.D.—6 months later consulted author because of prominence of O.D.—bruit subj. and obj.—swelling at inner canthus of eye and œdema—dil. of veins of conj.—no extraocular muscle palsies—fundus O.D. showed sl. œdema of disc—compression of swelling and inner canthus stops bruit—(V) O.D. = ⅓—no Wassermann—no X-ray—proposes (T) of KI, rest and gelatin injections.

(5) CARLOTTI.—Am. Ocul., Paris, 1908, vol. cxxxix and cxl, p. 450. M. 35, O.D. Trauma of head with unconsciousness—rt. 8th nerve palsy, earache and ecchymosis of lids—puls. exoph. and ptosis of O.D.—intracranial bruit reinforced in systole—dil. veins of conj. and retina—pupils inactive and dil. and ext. ophthalmoplegia O.D.—(V) O.D. = 5/10; O.S. = 5/7.5—visual fields normal—(T) rest gave no improvement—gelatin injections gave improvement.

(6) BECKER.—Jahresbericht Gesell. Natur. u Heilk., Dresden, 1908–1909, p. 1. M. 19, O.D. Revolver bullet entered rt. frontal region—2 days later exoph. and chemosis—amaurosis O.D.—pupil dil. and reactionless—enucleation 2 weeks after accident on mistaken diagnosis—2 months after operation author saw Pt. and found pulsation over rt. orbital fossa synchronous with radial pulse and bruit—no Wassermann—X-ray showed foreign body near sella—compression of rt. carotid shut off bruit.

(7) EVE.—Proc. Roy. Soc. Med., London, 1909–1910, vol. iii, Clin. Sect. 77. M. 39, O.S. After trauma headache, swelling O.S.—puls. exoph with systolic bruit over globe supraorbital and temporal regions—proptosis 8 mm.—enlargement of int. angular vein with thrill—fundus O.S. shows pulsation of dil. veins—diplopia—ophthalmoplegia in ext. O.S.—pupil reacts to light and darkness—good (V)—no Wassermann—X-ray shows fracture ?, left parietal region—(T) by rest and iodides—results not given.

(8) COTTERILL.—Edin. Med. Jour., 1910, N.S., vol. v, p. 54. M. 31, O.D. Trauma—great pain and loud subj. bruit—5th day after accident protosis—puls. exoph. with bruit heard all over rt. side of head—thrill over swelling about orbit—diplopia and rt. 6th nerve paralysis—fundus O.D. congested and haziness of both discs—(V) diminished O.D.—no X-ray—no Wassermann—compression of carotid stop pulsation—lig. of carotid followed by complete cure.

(9) FRY.—St. Barth. Hosp. Jour., 1910, vol. xviii, p. 40. M. 38, O.S. Fracture of skull with unconsciousness—3 months later puls. exoph. and proptosis—bruit widely disseminated—dil. mass of vessels at inner brow with thrill—dil. and tortuous veins of lid—chemosis—fundus O.D. shows dil. veins. (V) O.D. = 6/18; O.S. = 6/s. Pupil O.D. larger than O.S.—T. by rest and KI with marked improvement in exoph. and bruit.

(10) BALBUENA.—Arch. di. Oft. Hisp. Am., 1913, vol. xiii, p. 72. M. 25, C.S. Trauma. Shot in middle of forehead was followed by protrusion of O.S., puls. and proptosis—bruit loudest at upper inner angle of orbit—optic atrophy—T. by repeated gelatin injections subcutaneous and intravenous—cure of puls. exoph.

(11) MORETON.—St. Barth. Rep. Lond., 1914, vol. xlix, p. 73. M. Age ?, O.D. Trauma of head followed by pulsating exophthalmos with bruit—treated by rest, KI and low diet—improvement.

(12) *Ibid.*—F. 55, O.D. Two weeks after head trauma subj. bruit, frontal headaches and œdema of lids—puls. exoph. with proptosis, chemosis and systolic bruit best heard above eye and in temporal region—dil. of veins above eye—fundus shows dil. veins—paralysis of ext. ocular muscles and pupil dil. with no reaction to l.—wound tension—V. O.D. = 6/36 corrected to 6/18, O.S. = 6/6—compression of carotid did not stop bruit. Systematic digital compression gave improvement and lig. of int. carotid removed puls. exoph. but pallor of disc and poor V. was the end result.

(13) SATTLER.—Lancet Clinic, Cincinnati, 1914, vol. cix, p. 230. M. youth, Bilat. Blow on rt. side of head—unconscious—headache, vomiting and subj. bruit—exoph. O.D. with blurring of V.—one month later bilat. exoph. 2 cm. with puls. O.S. greater than O.D. proptosis and chemosis—bruit heard over O.S. and l. temple—fundi O.S. and O.D. showed dil. of veins and œdema of retina—marked dil. of epi bulbar veins—compression of l. carotid stops bruit and reduces exoph. T. not stated.

(14) TINDALL.—Hahnemann Monthly, Phil., 1915. vol. l, p 674. F. 55, Bilat. Trauma—bilat. puls. exoph. with bruit widely transmitted—dil. of veins of lids with puls.—fundi O.S. and O.D. showed engorgement of retinal veins with minute hemorrhages —not much visual disturbance—T. simply rest in bed and compress of eye—at end of 6 weeks much improved.

(15) RING.—Trans. Col. Physicians, Phil., 1916, vol. xxxviii, p. 338. F. 32, O.S. Trauma—History of congenital swelling at inner margin of l. orbit and following a second operation for this condition exoph. and proptosis of O.S. developed—with attack of influenza signs increased—puls. exoph. with bruit and chemosis and dil. of veins of lid and about eye, fundus O.S. showed areas of retina choroiditis—ocular movements sl. limited—V.—O.S. poor—T. not reported.

(16) RING.—Discussion of de Schweinitz case. Trans. Col. Phys., Phil., 1916, vol. lxxxvi, p. 343. Sex ?, Age ?, Side?. After trauma developed profound puls. exoph. and proptosis—2 months later lig. of common carotid was followed by almost complete recession of globe (improved).

(17) RHODES.—ANNALS OF SURGERY, 1916, vol. lxiii, p. 389. M. 27, O.S. Trauma resulted in severe headache and a subj. and obj. intracranial bruit and puls. exoph. 2 cm.—obj. bruit heard best over O.S. and l. temporal region—congestion of veins about eyes—pronounced chemosis—fundus O.S. shows marked venous stasis with enormous dil. and tortuosity and general œdema of retina—V.—O.S. blurred—no Wassermann nor X-ray—compression of l. carotid stops puls. and bruit.

(18) CAILLAUD.—Arch. d'opht. Paris, 1916–1917, vol. xxv, p. 746. M. 35, Bilat. Trauma followed in 19 days by puls. exoph. O.S. and then O.D.—bruit over O.S.—bilat. chemosis—dil. of veins above eyes with thrill O.S.—fundi showed dil. veins and arteries and pale discs—paresis of extraocular muscles more marked in O.S. than O.D.—corneal reflex about O.S. present O.D.—X-ray showed doubtful l. frontal fracture—no Wassermann—T. without avail by digital compression—lig. int. carotid both sides with 7-day interval—cure of bilat. puls. exoph.—hæmiplegia, aphasia and death 2 days post-op. Autopsy—no defect in cavernous sinus but int. carotid not examined.

(19) FRANKE.—Deutsche Med. Wchnschr. Leip. u. Berl., 1917, vol. xliii, p. 159. F. 40, O.D. Following trauma developed subj. bruit and puls. exoph.—obj. bruit also, systolic in time—paralysis of extraocular muscles, 3rd, 4th, and 6th nerves—pupil fixed—double lig. and removal of venous plexus behind eye—improved.

(20) SHAND.—Jour. Roy. Naval Med. Service, 1917, vol. cxi, p. 226. M. 34, O.D. Patient was struck on the head with wooden club—loss of consciousness for 20 min. with bleeding from nose and mouth—œdema about O.D.—10 days later pain and protrusion of O.D.—one month after accident bruit noticed and exoph. increased—fundus O.D. shows dil. retinal veins—unable to close lids O.D.—all movements of globe limited—blurring of vision—no Wassermann nor X-ray—compression of rt. carotid lessens bruit—lig. of rt. common carotid resulted in cure—sl. blurring of vision O.D.

(21) MAYOU.—Trans. Ophth. Soc. United Kingdom, 1917, vol. xxxvii, p. 186. M. age ?, O.D. Soldier received schrapnel wound behind rt. mastoid. X-ray located shrapnel in rt. temporal bone—puls. exoph. with bruit and proptosis—rt. facial palsy—lig. rt. int. carotid without avail.

(22) RIDLEY.—Discussion of Mayou's article (see above). Sex ?, Age ?, Side ?. Puls. exoph. with bruit developed after trauma of head—lig. of common carotid effected cure.

(23) WHITEHEAD.—Discussion of Mayou's article. Sex ?, Age ?, Side ?. Head trauma followed by puls. exoph. with intracranial bruit—lig. of common carotid effected cure.

(24) *Ibid.*—Sex ?, Age ?, Side ?. Puls. exoph. with intracranial bruit developed after head injury—lig. of common carotid was followed by cure.

(25) GALORINE.—Brit. Jour. of Ophth., March, 1918, p. 174. (Abstract.) M. 18, Side ?. Trauma puls. exoph. T. by lig. of superior ophthalmic vein—puls. exoph. receded satisfactorily but there was loss of V. and optic atrophy.

(26) *Ibid.*—Sex ?, Age 58, Side ?. Trauma puls. exoph.—lig. of sup. ophthalmic vein gave good results as regards exoph. and puls.

(27) *Ibid.*—M. Age ?, O.D. Head trauma was followed in 1 hr. by subj. bruit referred to rt. ear—puls. exoph. proptosis—complete ptosis—some loss of V.—lig. of rt. carotid and sup. ophthalmic vein followed by satisfactory results and dim. V. returned somewhat.

(28) GIBSON.—Med. Jour., Australia, 1918, vol. xi, p. 203. F. 19, O.S. Head trauma followed by puls. exoph. with loud continuous bruit accentuated during systole over O.S. and over l. temporal region—fundus shows dil. of veins—proptosis—V.—O.S. and O.D. 5/6—pressure over l. carotid stops puls. and bruit—lig. l. common carotid followed by improvement.

(29) SALUS.—Klin. Monatsbl. f. Augenh., 1918, vol. lx, p. 253. M. 22, Bilat. Following accident bleeding and blindness of O.D.—bilat. puls. exoph. O.D. = 25 mm., O.S. = 24 mm., widely disseminated subj. and obj. bruit—many dil. tortuous vessels of conj.—nodulated puls. swelling at inner canthi of both eyes—fundi O.D. shows retinitis proliferous traumatica O.S. dil. and tortuous veins, puls. arteries and hyperæmic papilla—marked limitation of extraocular movements O.D. less marked O.S.—pupil O.D. rigid O.S. reacts well—loss of sensation of cornea O.D.—V.—O.D. amaurosis, O.S. dim.—visual fields normal—X-ray shows bone defect in ant. fossa—disappearance of bruit upon rt. carotid compression—no report concerning T.

(30) BELL.—Arch. Ophth. N. Y., 1919, vol. xlviii, p. 84. M. 48, Bilat. Trauma of head followed by puls. exoph. O.D. = 24; O.S. = 22—proptosis—bruit heard in both temporal regions more on rt.—fundi show engorgement of retinal vessels O.S. and O.D. and choroid retinitis O.D.—diplopia and restriction in ext. ocular movements—V. O.D. = 20/50; O.S. = 20/40. X-ray reveals no signs of fracture—no Wassermann—lig. of common carotid followed by decided improvement.

(31) MARGARUCCI.—Riforma. Med. Nap., 1919, vol. xxxv, p. 919. M. Age ?, O.S. Trauma followed by puls. exoph. with intracranial bruit—1 year previous had lost opposite eye by war wound—dil. of vein about orbit and congestion of conj.—moderate chemosis—ptosis and deviation of O.S.—pupil reaction conserved—V. not impaired—Wassermann not taken—X-ray shows foreign body on rt. side in sphenoidal bone—compression of l. carotid does not stop bruit but pressure over rt. carotid does—conclusion was that arterio-venous communication was on l. side but failed to show because that eye had previously been enucleated—lig. of rt. common carotid was followed by some involuntary movements of l. side but no improvement of eye condition—following a second lig. of the same vessel the patient was cured.

(32) FASANO.—Discussion of Margarucci article. M. Age ?, Side ?. Fracture of the base of the petrous portion of the temporal bone a puls. exoph. resulted—no Wassermann—X-ray showed fracture—lig. of common carotid and jugular vein followed by death—fatality due not only to operation, but to severity of fractured skull. Autopsy—communication between cavernous sinus and int. carotid.

(33) MENACHO.—Arch. de Oft Hisp. Am., 1919, vol, xx, p. 20. Sex ?, Age ?, O.D. Trauma of rt. side of head was followed by puls. exoph.—intermittent systolic bruit—puls. swelling of veins in rt. temporal region—V. O.S. = 1/4, O.D.—blind—digital compression of artery was followed by disappearance of puls. and exoph. but atrophy of the optic nerve O.D. resulted.

(34) OURGAUD.—Marseille Med., 1919, vol. lvi, p. 607. M. Age ?, (soldier), O.S. Was knocked over by exploding shell and few days later developed exoph. which later pulsated—proptosis—bruit reinforced in systole was heard over eye—hyperæmia of coni

—fundus O.S. reveals dil. vein with undulations synchronous with pulse—no Wassermann nor X-ray— no T. nor outcome reported.

(35) BIELSCHOWSKY.—Munchen Med. Wchnschr., 1919, vol. lxvi, p. 700. M. 13, O.D. Four weeks after injury developed diplopia and subj. intracranial bruit—puls. exoph. O.D. 13 mm. with obj. bruit particularly over globe—puls. greatly dil. veins near inner canthus and of supraorbital region also of upper lid—dil. veins of conj.—fundus O.D. shows dilated and tortuous veins—complete 6th nerve paralysis O.D.—V. and visual fields normal—no Wassermann nor X-ray—T. digital compression of rt. carotid for 8 weeks results not given.

(36) LAPERSONNE and SENDRAL.—Bull. Acad. de Med. Par., 1919, vol. lxxxii, p. 515. M. 37, O.D. Trauma followed by non-puls. exoph. with subj. and obj. bruit widely transmitted and synchronous with pulse but best heard in rt. temporal and frontal regions—retinal veins dil. congested and tortuous—chemosis—no Wassermann—no X-ray —pressure over carotid stops bruit—lig. of rt. common carotid without preliminary course of digital compression effected cure.

(37) *Ibid.*—M. 36, Bilat. Exophthalmos following trauma with subj. and obj. continuous bruit best heard in temporal region and accentuated in systole—chemosis dil. puls. angular vein with thrill—complete ext. ophthalmophlegia with complete ptosis—V. poor—fundi show œdema of discs—no Wassermann—no X-ray—lig. of rt. common carotid gave neg. results—after 5 months of digital compression, lig. of left common carotid caused marked improvement.

(38) MAGEE.—Am. Jour. Ophth., 1919, vol. ii, p. 74. M. 24, O.S. After trauma puls. exoph. developed with proptosis and subj. and obj. bruit over l. temple—veins of upper lid dil. and tortuous—congestion of bulbar and palpebral conj.—severe headaches—fundus O.S. neg.—ptosis and complete immobility of globe—pupil somewhat dil. and reactionless to l.—photophobia—V. O.S. = ½—enucleation followed by lig. of int. carotid without a preliminary course of digital compression resulted in hæmiplegia—in a few weeks hæmiplegia cleared and eye condition was practically cured.

(39) ARGANARAZ and DEL VALLE.—Rev. Asoc. Med. Argent., 1919, vol. xxx, p. 377. M. 27, Bilat. Severe trauma to head and bleeding from nose and ears 9 mos. ago—subj. bruit and later violent pain of O.S. Bilat. pul. exoph.—34 mm. with bruit—thrill over dil. vein—dilatation of conj. vein—fundi showed congested and swollen discs—pupils dilated and fixed—cannot converge with either eye—X-ray neg.—no Wassermann—compression of carotid dim. bruit—lig. of int. carotid caused hæmiparesis which recovered in 5 months—puls. exoph. much improved.

(40) *Ibid.*—M. 33, O.D. Head trauma in auto accident—bleeding, loss of consciousness—deafness rt. ear—pul. exoph. O.D. and bruit—fundus O.D. optic neuritis—paralysis ext. ocular muscles O.D.—V. dim to 2 meters—no Wassermann—no X-ray—T. by subcutaneous injections of gelatin—cure.

(41) SPENCER.—Proc. Roy. Soc. Med. Lond., 1919–1920, vol. xiii, clin. sect., p. 73. M. 26, Bilat. Traumatic pulsating exophthalmos with subj. and obj. bruit best heard over O.S.—proptosis greater O.S. than O.D.—venous engorgement both upper lids, more on left—chemosis O.S.—6th nerve palsy O.S.—carotid compression stopped bruit—no Wassermann—X-ray neg. Lig. of left then rt. common carotid followed by slight improvement.

(42) GOMEZ, No. 2.—Brazil Med. Ros de Jan., 1920, vol. xxxiv, p. 29. Sex ?, Age ?, Side?. After trauma of head developed a puls. exoph. with intracranial bruit—lig. of common carotid brought "satisfactory results."

(43) DE RAFFELE.—Riforma Med. Napoli, 1920, vol. xxxvi, p. 345. M. 20, Side ?. Puls. exoph. of traumatic origin. Treatment not stated.

(44) THOMPSON.—Am. Jour. Ophth., 1920, vol. iii, p. 605. F. 32, O.D. Following head trauma sharp pain around O.D., then marked exoph. and systolic bruit—chemosis—enlarged vessels of upper lid—fundus O.D. showed veins full and tortuous and disc

margins indistinct—ocular movements limited with complete absence of abduction—pupil sl. dil. and reacts to l.—V. O.D. = 20/70; O.S. = 20/30; no Wassermann—nor X-ray. Compression of rt. common carotid stopped bruit—T. rest and digital compression—results not given.

(45) ERGGELET.—Munchen Med. Wchnschr., 1920, vol. lxvii, p. 412. M. Age ?, O.D. After trauma developed puls. exoph. with subj. and obj. bruit—extraocular palsies—pupil rigid—fundus O.D. showed only stubs of vessels—T. by lig. of int. carotid after lengthy preparation—improvement.

(46) POLLET and DECHERF.—Bull. et mem. Soc. de Chir. de Par., 1920, vol. xlvi, p. 102. M. 36, Bilat. Trauma of head with l. facial paralysis—15–20 days later continuous subj. bruit—puls. exoph. with chemosis of O.S. than of O.D.—obj. bruit over entire head—fundi showed dil. and tortuous veins—no X-ray—no Wassermann—compression of l. carotid stopped bruit—T. of digital compression failed—lig. of l. common carotid caused improvement.

(47) SATTLER.—Zeitschr. f. Augenh. Berl., 1920, vol. xliii, p. 534. M. 21, O.D. Fracture of skull followed by bruit in head and blindness O.D.—puls. exoph. 10 mm. with widely transmitted subj. and obj. intracranial bruit—puls. mass on upper lid and dil. of conj. vessels fundus O.D. showed puls. retinal veins—globe O.D. immobile—pupil moderately dil. and reactionless—tension O.D. 23–30 mm. O.S. 7–9 mm. V.—O.D. = 5/30; O.S. = 5/25—rest in bed and digital compression used without benefit—lig. of rt. int. carotid followed by improvement—3 months later developed rheumatism and died of pneumonia—no autopsy.

(48) *Ibid.*—M. 26, Bilat. Fractured skull followed by puls. exoph.—intracranial bruit and puls. swelling at inner angle of orbit—improvement with relapse in 1916—exoph. well marked again also puls. swelling near inner canthus—rt. pupil greater than l.—fundus O.D. showed dil. and tortuous veins—improved—2d relapse 1919, with exoph. of O.D. 19 mm. and now also of O.S. 18 mm.—puls. swelling enlarged—episcleral vessels dil. and tortuous and ciliary injection O.S. and O.S.—ext. rectus palsy O.D. pupil neg.—tension O.D. 15–11 mm., O.S. 14–15 mm.—fundi show dil. retinal veins and hemorrhages—medical T. and digital compression without avail—after lig. of rt. int. carotid pulsation returned in angular vein—and 3 days after lig. of angular vein bruit returned but became less marked, as did the other signs—improved.

(49) SATTLER.—M. 15, O.S. Fractured skull resulted in optic atrophy of O.S. and 4 months later exoph. then a puls. swelling at int. angle of orbit—17 years after accident, increase in exoph. and dim. motion of O.S.—then after one year of daily digital compression of carotid there was a sudden pain and signs of thrombosis of swelling at int. angle—following this marked improvement in signs—V.—O.D. = 5/5 corrected but disc of O.S. was atrophic.

(50) SATTLER.—Ztschr. f. Augenh. Berl., 1920, vol. xliii, p. 534. M. 17, O.D. One month after fractured skull bilat. 6th and 7th nerve palsies developed and 5 or 6 months later exoph. with blowing subj. bruit—proptosis 9 mm. forward 5 mm. downward—dil. veins with thrill in supraorbital region—fundus O.D. showed veins dil. and puls. and retinal hemorrhages—ocular tension O.D. 10–15 mm. O.S. 12–13 mm.—compression of carotid stopped bruit and puls.—course of digital compression for 5 days—lig. of int. carotid followed by improvement.

(51) SEYFORTH.—Munchen Med. Wchnschr., 1920, vol. lxvii, p. 1092. M. 22, O.D. Trauma followed by puls. exoph., proptosis and chemosis—bruit synchronous with pulse—dil. and tortuous veins of upper lid and in supraorbital region with thrill—no Wassermann nor X-ray—compression of carotid causes thrill to vanish—lig. of rt. int. and ext. carotid without preliminary digital compression and mass lig. of veins of orbital ridge—sepsis developed and death 9th post-op. day. Autopsy—communication between aneurism of int. carotid with cavernous sinus and also with dil. ophthalmic vein.

(52) POSEY.—Penn. Med. Jour., 1920, vol. xxiii, p. 658. M. 66, Bilat. After head trauma developed bilat. puls. exoph. (O.S. 6 mm. and O.D. less)—proptosis and subj. and obj. bruit—mass of dil. veins with thrill above both eyes—both eyes practically immobile; pupils 2½ mm. reacting sluggishly—fundi show marked dil. and tortuosity of veins and atrophy of both discs—compression of rt. carotid controlled bruit and puls.—partial occlusion by lig. of rt. carotid and lig. of prominent veins about eyes effected cure.

(53) *Ibid.* No. 2.—M. 50, O.S. Head trauma was followed by pain and swelling about O.S. and subj. bruit—puls. 6 mm.—dil. veins with thrill above eye—fundus shows dil. of veins and blurring of disc margins—only sl. ocular muscle palsy. V.—O.D. = 5/7.5; O.S. = 5/21; pressure over l. carotid stopped while pressure over rt. lessened bruit—no Wassermann and no X-ray—no preliminary course of digital compression of carotid. Hæmiplegia resulted after lig of l. carotid and bruit stopped—death 2 weeks later—no autopsy.

(54) *Ibid.* No. 3.—F. 43. Bilat. Followed head trauma developed subj. bruit and diplopia—bilat. puls. exoph. O.D. 24 mm; O.S. 22 mm.—bruit heard over both eyes—dil. blood-vessels in each supraorbital region—fundi show dil. and tortuous veins without neuritis—pupil O.D. 4 mm. with sluggish reaction while pupil O.S. 2½ mm. and active—rt. 6th nerve palsy—V. each eye 5/9. Pressure on l. carotid stopped bruit—lig. l. carotid and veins of rt. orbit and 10 days later resection of veins about O.S. resulted in improvement.

(55) FERRERO.—Arch. ital. di chir., Bologna, 1921, vol. iii, p. 405. F. 24, O.D. Fracture of skull in auto accident followed by pain in rt. temple and intermittent bruit in rt. ear—puls. exoph. O.D.—congestion about eye—diplopia—6th nerve palsy O.D.—no Wassermann—X-ray showed fracture of parietal and temporal bones probably extending into sella—lig. rt. int. carotid was followed by improvement.

(56) POULARD and BAILLIART.—Ann. d'Ocul. Par., 1921, vol. clviii, p. 227. Sex ?, Age ?, O.D. Trauma. After fracture of skull puls. exoph. appeared—both common carotids were lig. yet exoph. persisted and pupil remained dil. and immobile.

(57) YOAKAM.—Arch. Neur. and Psych., 1921, vol. v, pp. 754-755. M. 13, O.S. Gunshot wound. (See reference in present article, p. 16.)

(58) WEBER.—Abst. in Rev. Gen. d'Ophth., 1921, vol. xxxv, p. 140. M.—young man—O.S. Trauma. Bullet entered l. maxillary region and made its exit near rt. ear. Some weeks later a puls. exoph. developed. Lig. of common carotid resulted in improvement.

(59) UFFREDUZZI.—Giornale della R. acad. di Med. di Turino, 1921, vol. xxvii, p. 15. M. Age ?, Side?. Following temporo-parietal fracture developed puls. exoph.—lig. of int. carotid followed by cure.

(60) TEWFIK.—Bull. Ophthal. Soc. Egypt, Caro, 1921, p. 62. M. 15, O.D. Gunshot of head followed by puls. exoph. and proptosis—nodular swelling about eye with thrill—V.—O.D. = 6/18—no Wassermann—X-ray shows 2 small shot close to pituitary fossa—no T. nor results reported.

(61) MAY.—Jour. Iowa State Med. Soc., 1921, vol. xi, p. 346. M. 18, O.D. Fracture skull was followed by subj. bruit and puls. exoph.—obj. bruit heard best over globe—dil. veins at inner angle of orbit with thrill—engorged scleral veins—fundus O.D. shows dil. of vessels and obliteration of inf. retinal artery—bilat. int. squint—sl. dil. pupil which reacts to l & d—loss of sensation of cornea and about eye—V. corrected O.D. = 20/30; O.S. = 20/20—no Wassermann—X-ray shows fracture of base involving sphenoid—lig. of sup. ophthalmic vein was followed by cure.

(62) RIESE.—Deutsche Med. Wchnschr. Leip. u. Berl., 1921, vol. xlvii, p. 1090. M. 17, O.S.—Headaches and intracranial bruit after trauma—puls. exoph. with obj. bruit over it—dil. of veins of conj.—extraocular palsies O.S.—pupil dil. and reactionless—optic atrophy and loss of V.—no Wassermann nor X-ray—T. by systematic digital compression of carotid—no improvement—lig. of l. int. carotid gave improvement—later enucleation of O.S.

(63) HARTSHORNE.—Am. Jour. Ophth., 1921, 3. s., vol. iv, p. 353. F. 12, O.S. After head trauma marked exoph. (5 mm.) with obj. bruit over O.S. and l. temple—swelling about O.S.—fundus reveals dil. and tortuous veins—complete ptosis and paralysis of ext. ocular muscles O.S.—pupil dil. and inactive—V. = 15/15—fields normal—no Wassermann—X-ray neg.—bruit stopped by pressure over l. carotid—lig. of l. int. carotid resulted in almost entire disappearance of exoph. and improvement in ocular paralysis.

(64) COTTLE.—Am. Jour. Ophth., 1922, vol. v, p. 658. M. Age ?, O.S. Trauma—bullet penetrated cranial cavity entering left cheek—O.S. became swollen and V. was lost as well as all movements—puls. exoph.—proptosis—bruit widely transmitted—swelling in supraorbital region—conj. injected—corneal anæsthesia—hearing reduced on l.—X-ray shows bullet fragments in region of l. mastoid—lig. of l. common carotid very much dim. puls. exoph. and move[illegible]nts of globe [illegible]ned—partial return of sensation to eye—some puls. of globe when patient is in prone position.

(65) KEY.—Discussion of Wheeler's paper (see above). M. 49, Side ?. After head trauma puls. exoph. with bruit, p[illegible]ptosis and dim. of V. to 20/200—T. by almost constant digital compression for 10 days and end of this time bruit disappeared, proptosis receded and V. improved to 20/25—cure.

(66) *Ibid.*—Sex ?, Age ?, Side?. Severe head trauma followed by puls. exoph. and bruit but disappeared with use of Neff clamp on common carotid.

(67, 68) KING.—Discussion of Wheeler's paper. Sex ?, Age ?, Side ?. Two cases of trauma—puls. exoph. treated by placing a silver band on the carotid arteries only partially obliterating the lumen—in 2 weeks after the first operation to the arteries will be completely obliterated.

(69) KUNZ.—Zeit. fur Augenh., 1922, vol. xlvii, p. 166. M. 19, O.S. Trauma. Two months after head injury with symptoms of fractured skull pt. developed puls. exoph.—4 months after injury lig. of l. int. carotid caused symptoms to disappear—6 months later puls. exoph. returned.

(70) HEUSER.—Discussion of Kunz's paper (see above). Sex ?, Age ?, Side ?. Trauma following fracture of base of skull—puls. exoph. with bruit—T. not described.

(71) FENTON.—Am. Jour. Ophth., 1922, vol. v, p. 802. F. 24, Bilat. Blow on jaw—unconsciousness—on awakening heard bruit in rt. ear—one week later diplopia and extra-ocular palsies—1 month later puls. exoph. O.D. with obj. bruit—then paresis of 3rd, 4th and 5th nerves and corneal ulcer—5 months after injury bilat. puls. exoph. and paralysis of extraocular muscles on both sides—no Wassermann nor X-ray—lig. of rt. int. carotid (8 months) caused marked improvement.

(72) CAUCHOIX.—Bull. et Mem. Soc. de Chir. de Par., 1921, vol. xlvii, p. 153, *loc. cit.*, 1922, vol. xlviii, p. 20. M. 47, O.D. Trauma—27 days later puls. exoph. O.D.—bruit accentuated in systole heard best over O.D.—dil. of veins about O.D.—no Wassermann—compression of rt. carotid stopped these signs—lig. of rt. and l. common carotid arteries, 3-day interval—improvement but recurrence in 8 months—lig. of rt. sup. ophthalmic V.—no report of result.

(73) SWIFT.—Am. Jour. Ophth., 1921, 3. s., vol. iv, p. 124. M. 23, Bilat. Traumatic—bilat. puls. exoph. with subj. and obj. bruit heard in temporal and frontal regions—fundi show engorgement and tortuosity of arteries and veins and paleness of both discs—bilat. ptosis and both eyeballs fixed and sl. divergent—pupils dil. and reactionless and marked loss of V. in both eyes—no Wassermann nor X-ray—lig. of rt. int. carotid caused marked improvement of O.D. and sl. improvement O.S.

(74) PASCALE.—Ann. ital. di. chir. Nap., 1922, vol. i, p. 144. M. 20, O.S. Shot near l. ear—subj. bruit of l. side of head and l. facial paralysis—puls. exoph. with obj. bruit reinforced in systole—dil. of small arteries and veins of conj.—fundus O.S. sl. hyperæmia and dil. of veins—no Wassermann nor X-ray—V. O.D. = 9/10; O.S. = 1—urine normal—compression of carotid checks bruit—lig. of l. int. carotid effected complete cure.

(75) *Ibid.*—M. 17, O.S. After being kicked in region of O.S. by a mule the local condition subsided except for œdema of upper lid—subj. intracranial bruit—auscultation revealed systolic bruit—puls. exoph.—tumefaction of upper eyelid with visible puls. and palpable thrill—no Wassermann—1 year later lig. of common carotid—death. Autopsy: Arterio-venous aneurism of int. carotid.

(76) WHITHAM.—Am. Jour. Ophth., 1923, vol. vi, p. 81. M. 30, O.S. Fracture of skull followed by rt. hæmiplegia and aphasia which soon cleared—marked puls. exoph. with subj. and obj. bruit—chemosis and great engorgement of palpebral and bulbar conj.—fundus O.S. shows retinal veins dark and dilated and disc margins blurred—lagophthalmos and definite restriction of movements of O.S.—normal pupil reactions—V. O.S. = 20/60; O.D. = 20/20 + + —Visual fields neg. Wassermann neg.—X-ray showed linear fracture of l. orbit—lig. l. int. carotid followed by great improvement.

(77) *Ibid.* No. 3.—M. 49, O.S. Six days after head trauma developed bilat. prominence of eye and congestion—l. subtemporal decompression was followed by aphasia and onset of puls. exoph. of O.S. with widely transmitted bruit—chemosis—dil. of retinal veins—cloudiness of media and cornea—6 weeks later lig. of l. int. carotid followed by complete cure.

(78) *Ibid.*, No. 4.—F. Age ?, O.S. One week after trauma developed puls. exoph. O.S. with loud bruit over globe and over l. side of head—œdema of bulbar conjunctivum fundus O.S. revealed full and tortuous veins and paleness of optic nerve—limitation of movements of globe—pupil neg. V. O.S. = 20/30; O.D. = 20/20—no Wassermann—X-ray neg.—lig. of l. int. carotid gave improvement.

(79) *Ibid.*, No. 5.—M. 45, O.S. Three weeks after head trauma diplopia, severe pain and puls. exoph. (3 mm.) of O.S. appeared—continuous intracranial bruit accentuated in systole, also thrill—œdema of lids and of bulbar conj.—with dil. of vessels—all movements of eye absent—pupil reacts to i and d—V. O.D. = 20/30; O.S. = 6/200—5 days later lig. of l. ext. carotid and metal band placed on l. common carotid—patient discharged cured on 16th post-operative day.

(80) SANTA CECILIA.—Brazil Med. Rio de Jan., 1923, vol. xxxvii, p. 4. M. 19, O.D. Trauma followed by puls. exoph. synchronous with pulse with subj. and obj. bruit—conj. O.D. congested—fundus O.D. showed engorgement of veins—diplopia and rt. 6th nerve palsy—lig. of rt. common carotid without avail but later realization with lig. of ext. carotid gave marked improvement.

(81) Case report in present article, 1923.

(82) Case report in present article, 1923.

(83) Case report in present article, 1923.

CASES WITH ETIOLOGY NOT STATED

(1) COVER and STEVENS.—Ophth. Rec., 1909, vol. xviii, p. 128. Sex ?, Age ?, Etiol. ?. Puls. exoph. was cured by ligation of the common carotid.

(2) FULLER.—Discussion of Halstead's paper. Surg., Gyn. and Obs., 1911, vol. xii, p. 298. Sex ?, Age ?, Side ?, Type ?. Pulsating exophthalmos—ligation of common carotid with improvement and the recurrence—later pressure over orbit for long intervals effected cure.

(3) HEPBURN.—Am. Jour. Ophth., 1920, 3. s., vol. iii, p. 144.—M. Age ?, Side ?, Type ?. Pulsating exophthalmos with proptosis and bruit—dilated supraorbital veins—vision 6/6—ligature of common carotid did not improve the condition.

(4) ROBINEAU.—Gaz. des Hosp., 1921, vol. xciv, p. 187. Sex ?, Age ?, Side ?, Etiol. ?. Puls. exoph. in which ligation of one common carotid was made and then 5 or 6 weeks later the other common carotid was ligatured—the results were satisfactory.

(5) BURCI.—Discussion of Margarucci article (see above). M. Age ?, Side ?, Etiol. ?. Pulsating exophthalmos cured by ligation of internal carotid.

(6) KEY.—Discussion of Wheeler's paper (see above). Sex ?, Age ?, Side ?, Type ?. Case of pulsating exophthalmos with bruit—proptosis and some loss of vision—after ligature of the common carotid signs disappeared and vision improved (cure).

(7) MANN.—Discussion of Becker's case (see above). M. Age ?, Side ?, Etiol. ?. Pulsating exophthalmos with bruit was *cured* after an accidental severe head bumping.

(8) CLEGG.—Discussion of Mayou's article (see above). M. Age ?, Side ?, Type ?. Puls. exoph. without subjective bruit. Treated by ligation of carotid and patient when seen 1 year later was entirely cured.

(9) ELLIOT.—Discussion of Mayou's article. Sex ?, Age ?, Side ?, Type ?, Puls. exophthalmos with bruit heard all over head—patient improved without treatment.

(10) COLLINS.—Discussion of Mayou's article.—M. Age ?, Side ?, Etiol ?. Pulsating exophthalmos with intracranial bruit—ligation of the common carotid was followed by disappearance of the pulsating exophthalmos and then some return later (improved).

BOOK REVIEWS

DISLOCATIONS AND JOINT-FRACTURES. By FREDERIC J. COTTON, M.D., Visiting Surgeon to the Boston City Hospital. Second Edition, Reset, 8vo, 745 pages with 1329 illustrations from drawings by the author. Philadelphia and London: W. B. Saunders Company, 1924.

Doctor Cotton first embodied his experiences and opinions in the matter of dislocations and joint-fractures in his book which was published fourteen years ago. That book was sufficient to establish his reputation as an accurate observer and an original thinker. Still more will this new edition add to his reputation. Much has happened since 1910. Experience has broadened—judgment has matured—surgery has developed, and it is well that the volume of so many years ago be now revised.

The special value for this book rests in its personal character. There is a certain dogmatism and positiveness about the way in which the writer's views are presented which is attractive and impressive. The field to which the author proposes to confine himself is the one in which most of the difficulties of traumatic bone surgery are found.

We recommend to the reader the careful perusal of the introduction which in itself, in the view which it gives of the problems connected with joint-fractures, is full of real surgical philosophy. We endorse especially this statement of his: "No damage to any machinery, human or other, increases its efficiency, and in the human machinery we cannot replace parts. Many breaks and dislocations do damage absolutely irreparable; many do damage entirely unrecognizable at the time. * * * Our duty is to obtain, in the given case, the best results obtainable in this case by whatever means are at hand. * * * No book can make a man a fracture expert. * * * Experience, common sense, capable and trained fingers, and perhaps most important of all what you call mechanical sense, must help him out."

The reviewer would like to emphasize this statement in fracture work. Common sense and mechanical sense are essential to make experience and technical training of especial value in fracture work. It is in the degree to which this book reflects these two elements, common sense and mechanical sense, that it is to be commended.

The wealth of illustrations which the book contains will increase its value to the student. The teachings are sound and clearly and forcibly presented. It is a welcome addition to the literature of joint and bone traumatisms in which American surgery is already so rich. LEWIS S. PILCHER.

OPERATIVE SURGERY. By J. SHELTON HORSLEY, M.D., St. Louis. C. V. Mosby Company, 1921.

This single volume of 721 pages, containing 613 original illustrations devoted to operative surgery, is largely the result of the author's own personal experience. There is no attempt made to describe all surgical operations.

The ones that are described, however, have either been done by the author, himself, or are those, which, in his opinion, are best suited for the particular condition under discussion. The work while not complete is comprehensive. As a rule only one method is described. In several instances, however, several procedures are given. The sections on blood-vessel surgery, plastic surgery, gastro-intestinal surgery, genito-urinary surgery, and the chapters dealing with technic, drainage, infection, complications following operations, hemorrhage, shock and blood transfusion are especially illuminating and instructive.

The outstanding feature of the work seems to be the emphasis placed upon the importance of the physiologic function and the interpretation of the biologic processes that follow surgical operations. For instance, a cigarette drain placed into an abscess cavity does indeed accomplish drainage, but not mechanically. It drains because it is sufficiently irritating to produce a stimulus which reverses the flow of the local lymphatic circulation. The lymph instead of absorbing the pus pours out into and around the drain in an attempt to extrude it. Plating of fractures gives good primary results from a mechanical standpoint, but the biologic results are far from ideal.

The book under review is certainly not a text-book for the use of medical undergraduates. But, in the library of the younger doctor, who is studying to become a surgeon; to the surgeon who has already arrived; to anyone interested in the technic of operative surgery, this book will find a welcome place.

MERRILL N. FOOTE.

MODERN UROLOGY, in original contributions by American authors, edited by HUGH CABOT, M.D. 2 vols., 8vo, 2nd and revised edition. Lea and Febiger, Philadelphia, 1924.

This work is written by twenty-eight of the leading urological surgeons of the United States. It is by far the best book that has ever been written on the subject up to date. Most of the chapters are complete in every detail, giving full references to original sources, which is a most essential detail for a book of its sort.

Those articles which stand out particularly in vol. i, are as follows: IV.—Syphilis of Genito-urinary Organs. V.—Anatomy, Anomalies and Injuries of the Penis. VIII.—Infections of Urethra and Prostate. XIII.—Hydrocele, Hæmatocele, Spermatocele and Varicocele. XVI.—Tumors of the Testicle. XVII, XVIII, XIX on the Prostate Gland.

The chapter on prostatic obstruction was written in the previous edition by the inimitable and beloved, the late Dr. Paul M. Pilcher, and will always stand as a monument to his genius and thoroughness. In the present edition this subject is ably handled in an unbiased manner, bringing the subject up to date.

If there are any weaknesses in this volume, they are in Chapter I, which is not complete in that it does not describe nearly all the valuable instruments of the day. Chapter III is so poorly illustrated that it is almost spoiled and Chapter VI is merely an exposition of the author's personal views, which does not seem to be sufficient for a book of this character.

The outstanding thesis in the second volume is Chapter VIII.—Anatomy, abnormalities, injuries and diseases of the ureter. The author has presented the subject in a masterful manner. It is beautifully illustrated and should be a permanent monument to his scientific ability and personal pertinacity. The subjects particularly well handled are: Chapter II.—Malformations and diverticula of bladder; Chapter IV.—Infections of the bladder; Chapter V.—Stone in the bladder; Chapter VII.—Tumors of the bladder; Chapter XII.—Infections of the kidney. In fact, all the articles in the second volume are masterpieces with the possible exception of those dealing with parasites of the bladder, which present a very difficult subject and concerning which nothing particularly new has been contributed of late years.

In reviewing a work of such excellence, one hesitates to make any criticism of an adverse nature whatever. There are one or two points which, if corrected, might strengthen the work as a whole.

It is unfortunate that space prevented the publication of the historical review of Genito-urinary Surgery in America, as this is always a most interesting feature of any book.

Blood chemistry is very lightly handled for such an important subject and the question of local and regional anæsthesia, which has assumed so much importance of late, is merely mentioned.

These two volumes must necessarily be used by the special student in urology and not by the general student in medicine and surgery, therefore the author who completed his thesis without giving full and complete references to all of his original sources of information has not presented a finished product for this type of publication. When one observes the large number of authors involved, he notices that the list of incomplete chapters is happily very small.

OSWALD S. LOWSLEY.

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